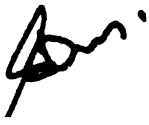
Diseases of the Eye



nerve cells situated near the middle line in the floor of the aqueduct of Sylvius beneath the corpora quadrigemina or colliculi (Figs. 290—293). The cells nearest the middle line towards the anterior part of the third nucleus are smaller than the others; they, with the cells of the opposite side, form an unpaired nucleus with two divergent horn-like processes in front (the Edinger-Westphal nucleus) (Fig. 294), which probably supplies fibres to the ciliary muscle (accommodation) and sphincter iridis (constriction of the pupil). It is probable that in the great large-celled lateral nucleus the levator palpebræ is represented most anteriorly, then from before backwards elevation of the eye, adduction, and depression, while abduction is relegated to the sixth nucleus, much farther back in the medulla (Fig. 294). There is little decussation of the fibres from the third nuclei of the two sides in the anterior part, but a considerable amount in the posterior part.

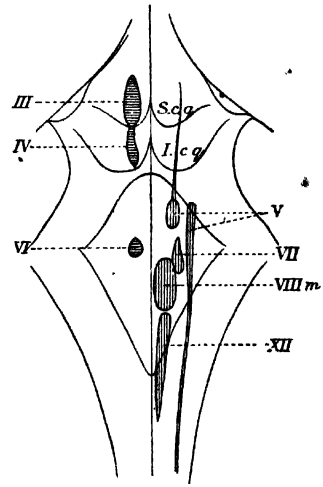



FIG. 290.—Diagram of the positions of the nuclei of the third, fourth, and sixth nerves, seen from above. *S.c.q.*, superior corpus quadrigeminum; *I.c.q.*, inferior corpus quadrigeminum.

The fourth nerve is unique among motor nerves in having a dorsal decussation. Nearly, if not quite, all the fibres decussate in the superior medullary velum and are distributed to the superior oblique muscle of the opposite side.

The sixth nucleus is in the immediate vicinity of the facial (seventh) nucleus (Figs. 290, 293), the fibres from which make a large bend around it (Fig. 295). Hence vascular and other lesions of the sixth nucleus are very liable to be accompanied by facial paralysis on the same side. All the fibres of the sixth nerve are distributed to the external rectus of the same side.

The peculiarities of distribution of the fibres from the third,

fourth and sixth nuclei to muscles partly on one side and partly on the opposite side of the body show that the nervous mechanism of co-ordination of these muscles is extremely complex.

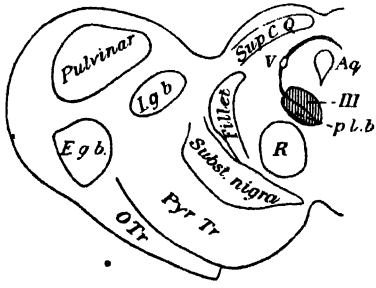



FIG. 291.—Diagram of transverse section of the mesencephalon at the level of the third nucleus (level of 1, Fig. 296). *Sup. c.q.*, superior corpus quadrigeminum; *R*, red nucleus; *l.g.b.*, internal geniculate body; *E.g.b.*, external geniculate body; *O Tr.*, optic tract.

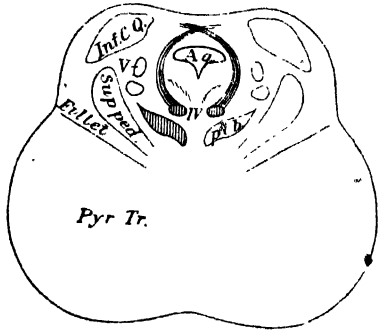



FIG. 292.—Diagram of transverse section of the mesencephalon at the level of the fourth nucleus (level of 2, Fig. 296). *Aq.*, aqueduct of Sylvius; *Inf. c.q.*, inferior corpus quadrigeminum; *p.l.b.*, posterior longitudinal bundle; *Sup. ped.*, superior peduncle of the cerebellum; *Pyr. Tr.*, pyramidal tract.

A large and important tract of nerve fibres, derived in part from the anterior columns of the spinal cord, lies below and close to the third, fourth and sixth nuclei. This is the posterior longitudinal bundle (Figs. 291—293, 296). Fibres pass between it and the nuclei under consideration; they probably have important functions in the co-ordination of movements and equilibration, which are so intimately related with vision. Among these fibres are also some which link up the sixth nucleus of one side with the third nucleus of the other in some such manner as depicted in Fig. 296, though the exact course of the fibres has not been definitely proved. These fibres are concerned in conjugate deviation of the eyes to one or other side. Hence when one sixth nucleus is destroyed the patient is unable to turn his eyes to the same side, though the power of convergence is unimpaired. Nuclear sixth nerve paralysis therefore causes loss of conjugate deviation of the eyes to the same side, and is very likely to be associated with facial

paralysis on the same side, whereas peripheral sixth nerve paralysis causes only loss of power of movement of the same eye to the same side.

The student should revise his knowledge of the anatomical relations of these and the neighbouring cranial nerves in their course from the nuclei to their respective terminations.

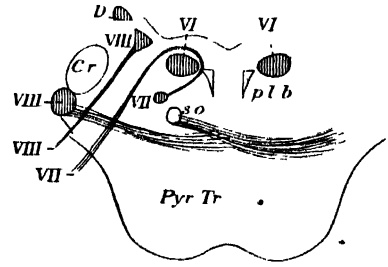



FIG. 293. Diagram of transverse section of the pons at the level of the sixth nucleus (level of 3, Fig. 296). *p.l.b.*, posterior longitudinal bundle; *D*, Deiter's nucleus; *s.o.*, superior olive; *C.r.*, restiform body; *Pyr. Tr.*, pyramidal tract.

**Orientation.** Orientation of objects in space depends upon their relation to the nodal point of the eye, *i.e.*, the position of an object is determined by the line passing through the object and the nodal point, the spot where this line cuts the retina being the position of the retinal image of the object. Conversely an object is said to be *projected* along the line joining the retinal image

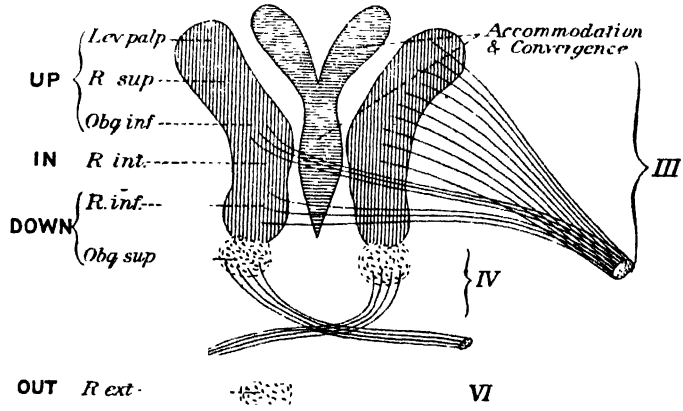



FIG. 294.—Diagram of the probable position of the nuclei of origin of the fibres to the ocular muscles in the third, fourth and sixth nuclei.

with the nodal point. Objective orientation determines the relative positions of objects to each other. Subjective orientation, or the exact relation of the situations of objects

to ourselves, is much more complex, depending upon an accurate knowledge of the position of the body and of the eyes in the body, derived largely from the muscular sense.

**Corresponding Points.** When a distant object is looked at the visual axes are practically parallel: the object forms an image upon each fovea centralis. An object to one side of the object looked at forms its retinal images upon the temporal side of one retina and upon the nasal side of the other; these are called corresponding points. Points on the two retinæ which are not corresponding points in this sense of the term are called *disparate points.* If an object forms its retinal images upon disparate points it will be seen double (binocular diplopia). If the disparity is slight there is a great tendency to move the eyes so that the images may be fused. It will be noticed that the two foveæ are corresponding points.

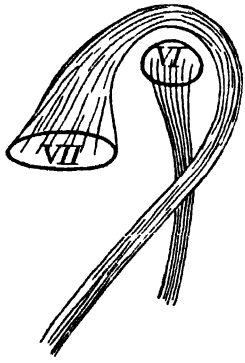



FIG. 295.—Diagram of the sixth nucleus and its relation to the nucleus and the emerging fibres of the seventh nerve. The relations cannot be accurately depicted in one plane.

When a near object is looked at the eyes converge the requisite amount to bring the two retinal images of the object upon the two yellow spots.

**Binocular Vision.** When the eyes are normal the individual sees clearly with both eyes the object looked at. The retinal images of the two eyes are not, however, identical. This is obvious when it is remembered that there is a considerable distance between the two eyes. If the object is a solid body, *e.g.*, a cube, the right eye sees a little more of the right side of the object, and *vice versâ*. The two images are fused psychologically, and it is this fusion of the slightly diverse images, combined with other facts derived from experience, which enables the person to appreciate the solidity of objects. The estimation of the relative distance of objects in or near the line of vision is still more complex. It probably depends upon the fact that the images of objects farther or nearer than the object fixed are situated at disparate points on the retinæ. A more distant object will produce heteronymous diplopia, a nearer homonymous diplopia, as can be shown by experiment. The diplopia is suppressed in actual vision, but it produces a

psychological impression which is translated into appreciation of distance. It will suffice if it is well understood that accuracy of stereoscopic and topical vision depends upon good sight with both eyes simultaneously.

**Convergence and Accommodation.** When a distant object is observed by an emmetropic person the visual axes are parallel and no effort of accommodation is made. If a near object is observed the eyes converge upon it and an effort of accommodation corresponding with the distance of the object is made. Convergence can be tested roughly by making the patient fix a finger or pencil which is gradually brought nearer to the eyes in the middle line. The eyes should be able to maintain convergence when the object is 8 cm. ($3\frac{1}{2}$ inches) from the eyes. If outward deviation of one eye occurs before this point is reached the power of convergence is deficient. There are various methods of recording the amount of convergence. One very convenient method employs the *metre angle* as a unit. Suppose an object to be situated in the median line between the two eyes at a distance of one metre from them. Then the angle which the line joining the object with the centre of rotation of either eye makes with the median line is called one metre angle (Fig. 297). With an interpupillary distance of 60 mm. this angle is about 2°. If the object is two metres away the angle is approximately half as great, or $\frac{1}{2}$ m.a. If the object is 50 cm. away the angle will be 2 m.a. Now, the amount of accommodation which an emmetropic eye exercises in order to see clearly an object 1 m. away is 1 D, 2 m. away 0·5 D, 50 cm. away 2 D, &c. Hence with an emmetropic person the amount of convergence, reckoned in metre angles, is the same as the amount of accommodation reckoned in dioptres.

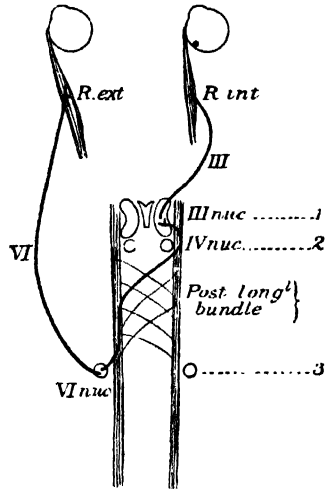



FIG. 296.—Diagram of the course of the fibres from the sixth nucleus which are concerned in conjugate deviation of the eyes. 1, 2, 3, lines of section of Figs. 291–293.

The amount of convergence can also be measured by prisms.

If an object one metre distant is looked at through a prism with the base directed outwards placed before one eye, *e.g.*, the right, it may still be seen as a single object (Fig. 298). Now in order that the object may form its image upon the fovea of this eye it is necessary that the eye should be turned inwards an amount corresponding with the angle of deviation of the prism (*vide* p. 28). This method, besides affording a method of recording amounts of convergence, also shows that the relationship between convergence and accommodation is somewhat elastic. In the experiment described, although the amount of accommodation exercised remains the same, the amount of convergence is altered. Indeed, if the relationship were quite unalterable a hypermetropic person would invariably have diplopia, for his accommodation is always in excess of the corresponding value of the amount of convergence exerted by an emmetrope. Moreover, the power to converge would gradually be lost *pari passu* with loss of accommodation in advancing age.

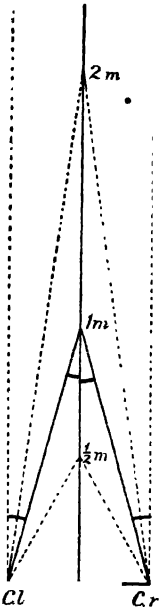
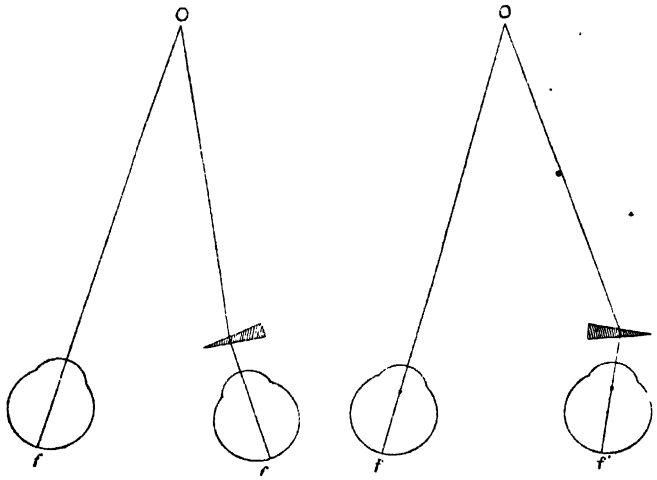



FIG. 297.—Diagram of the metre angle. *Cr.*, *Cl.*, centres of rotation of the right and left eyes.

If in the experiment described above the prism is held before the right eye with its base inwards it will still be found possible to see the object single (Fig. 299). Further, a distant object is still seen single under the same conditions if the prism has only a small angle of deviation. This can only be accomplished by active divergence of the eyes to an amount corresponding with the angle of deviation of the prism. The power of divergence, which may therefore be considered to be negative convergence, is much less than the power of convergence. Just as the difference in the amount of accommodation between the far point and the near point is called the amplitude of accommodation, so the difference in convergence between the far point and the near point is called the *amplitude of convergence*. Whereas, however, negative accommodation, *i.e.*, ability to see a point beyond infinity, or in less mathematical terms, so to flatten the lens that a myope could see clearly without glasses, is impossible, negative convergence, as has been seen, is possible

within small limits. The amplitude of convergence therefore consists of a negative portion and a positive portion. The former is measured by the strongest prism, base inwards, which can be borne without producing diplopia in distant vision. The latter is measured by the strongest prism, base




FIGS. 298-299.—Diagrams of the action of adducting and abducting prisms. O, object of fixation; $f, f'$, left and right foveæ centrales.

outwards, which can be borne without producing diplopia in the nearest possible vision.

The convergence synkinesis is so co-ordinated that the energy exerted is accurately divided between the two internal recti. Hence it is found that the effect is the same in the above experiments whether the prism is placed before only one eye, or a prism of half the strength is placed before each eye.

# CHAPTER XXVII

## Paralytic and Kinetic Strabismus Synkineses Nystagmus

**Strabismus** (στρέφειν, to turn) or *squint* is a generic term applied to all those conditions in which the visual axes assume a position relative to each other different from that required by the physiological conditions. Strabismus may be provisionally divided into two great groups: (1) those due to known cause; (2) those due to unknown cause. To the first group belong (*a*) those due to paresis or paralysis of one or more of the extrinsic muscles—*paralytic strabismus;* (*b*) those due to irregular activity or over-activity of individual muscles or groups of muscles—a sub-group which I propose to designate *kinetic strabismus*. To the second group belong (*a*) those cases which are characterised by the fact that the visual axes, though abnormally directed, retain their relative position in all movements of the eyes; they are therefore termed *concomitant* or *comitant strabismus*. Another sub-group of the second class is (*b*) cases in which there is *latent strabismus* or *heterophoria*.

### Paralytic Strabismus

**Signs and Symptoms.** (1) *Limitation of Movement.* In paralysis of an ocular muscle the ability to turn the eye in the direction of the normal action of the muscle is diminished or lost. In slight paresis the defect in mobility may be so small as to escape observation without special tests. In all positions in which the affected muscle is not brought actively into play the visual axes assume their normal relationship.

Limitation of movement is tested roughly by fixing the patient's head and telling him to follow the movements of the surgeon's finger. The finger should be held vertical in testing horizontal movements, horizontal in testing vertical movements. An accurate record of the movements of each eye can be obtained by taking the field of fixation. The patient is seated at the perimeter as for recording the field of vision. With the head fixed and the other eye screened the patient

looks as far as possible along the arc of the perimeter, test types being moved in from the periphery until he is just able to read them. The normal field of fixation is about 50° downwards and 45° in all other directions.

When the eyes are turned in the direction of the normal action of the paralysed muscle the affected eye remains stationary. It deviates therefore relatively to the other eye; this position is called the *primary deviation* (Fig. 300).

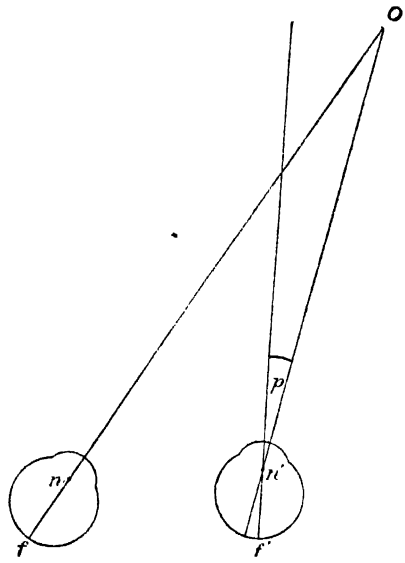



FIG. 300.—Diagram of primary deviation in paresis of the right external rectus. *p*, angle of primary deviation; *n*, *n'*, left and right nodal points.

The angle of deviation is the angle which the line joining the object observed with the nodal point makes with the visual line.

If the sound eye is covered by a screen, and an attempt is made to fix an object so situated that the paralysed muscle is brought into play, it will be found that the eye behind the screen deviates more than the primary deviation of the paralysed eye. For example, if the right external rectus is paralysed and the left eye is covered, then on attempting to fix an object situated to the right with the right eye the left eye will deviate very much to the right, so much in fact that

its line of vision is well to the right of the object fixed. Hence, if the screen is removed suddenly the left eye will spring back to the left so as to take up fixation. This deviation of the sound eye is called the *secondary deviation* (Fig. 301). The reason why the secondary deviation is greater than the primary is that in conjugate deviation of the eyes the nervous energy is equally distributed between the muscles of the two

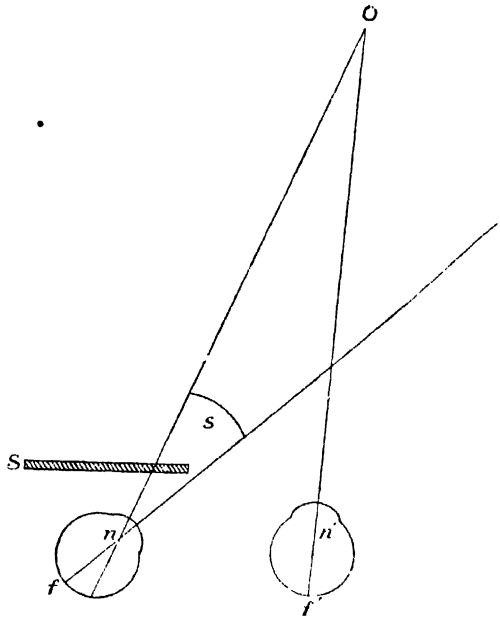



FIG. 301.— Diagram of secondary deviation in paresis of the right external rectus. *S*, screen in front of left eye; *s*, angle of secondary deviation.

eyes. Now the effort to take up fixation with the paralysed or paresed eye is much greater than normal. Consequently the sound eye behind the screen moves through a greater distance than normal, *i.e.*, through a distance corresponding with the excessive effort exerted. This feature is of great importance because when well marked it distinguishes paralytic squint from the concomitant type in which the secondary deviation is equal to the primary.

(2) *Diplopia.* The chief complaint of patients with paralysis of an extrinsic muscle is often that they see double. Diplopia

occurs only over that part of the field of fixation towards which the affected muscle or muscles move the eye. If both eyes are functional and one deviates, *i.e.*, if the visual axes are not parallel in looking at a distant object, or if the amount of convergence is not accurately adapted to the position of the object in near vision, *binocular diplopia* results. When the deviation is due to paralysis of one or more extrinsic muscles, the eye on the sound side fixes the object accurately, while the other eye deviates. Suppose the left eye fixes accurately while the right deviates inwards, a bright, sharply defined foveal image is seen with the left eye. The image formed by the object on the right retina, falling as it does upon the line joining the nodal point with the object, lies to the nasal side of the retina. The patient being unconscious of the malposition of his eye orients the object subjectively as if the eye were straight. He knows from experience that objects which form their images upon the nasal side of the retina are situated to the temporal side. He therefore projects the object with this eye to the right of its actual position. This is called *homonymous diplopia*, because the object as seen by the right eye is to the right of the object as seen by the left eye (Fig. 302).

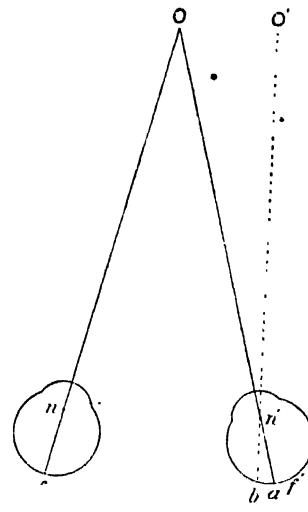



FIG. 302.—Diagram of homonymous diplopia. *f*, *f'*, left and right foveæ; *n*, *n'*, left and right nodal points. The image of O formed at *a* is projected as if *a* were the fovea, *i.e.*, to O'.

If the right eye deviates outwards, *heteronymous* or *crossed diplopia* results, because the object as seen with the left eye lies apparently to the right of the object as seen by the right eye (Fig. 303).

In binocular diplopia the image seen by the squinting eye (false or apparent image) is less distinct than that seen by the fixing eye (true image), because only in the latter case does the image fall upon the fovea centralis. The angular displacement of the false image is equal to the angle of deviation of the eye.

(3) *False Orientation.* It will be seen from what has

already been said that false orientation is a necessary accompaniment of binocular diplopia. Suppose that a patient whose right external rectus is paralysed shuts his left eye and attempts to fix an object situated towards the right. Let him now quickly strike at the object with his extended index finger. The finger will pass considerably to the right of the object. This is called *false projection*. It depends upon exactly the same principle as the increase of the secondary deviation. The object is projected according to the amount of nervous energy exerted; as this is greater than that exerted under normal circumstances, the object is projected too far in the direction of action of the paralysed muscle. It is essential that the finger should be directed at the object quickly, otherwise the error is noticed and compensated for. For example, if under the same circumstances the patient is told to walk towards an object situated at some distance to the right, he first steps too far to the right, then recognises his mistake and corrects it. In old paralysis the patient may learn by experience completely to compensate for the deficiency.

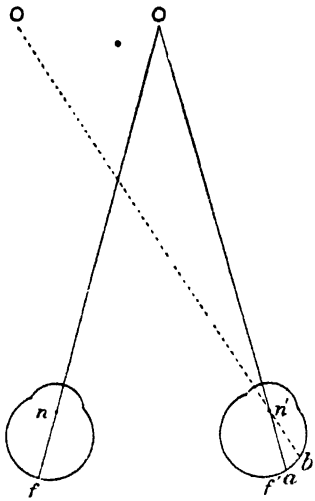


FIG. 303.—Diagram of heteronymous (crossed) diplopia.

(4) *Position of the Head.* The patient holds his head so that his face is turned in the direction of action of the paralysed muscle. For example, in paralysis of the right external rectus the patient keeps his head turned to the right. The object of this manœuvre is to abolish the diplopia and its attendant unpleasant consequences as much as possible. In complex paralysis the position of the head is still such as to relieve the diplopia to the maximum extent, the position being adopted unconsciously.

"*Ocular torticollis*" is a term sometimes applied to tilting of the head to compensate defective vertical movements of one eye. It is distinguished from true torticollis in that there is a simple tilting of the head, the chin not being rotated towards the opposite shoulder; moreover, the sterno-mastoid is not unduly contracted.

It occurs chiefly in cases of congenital origin—probably malinsertion of the muscles—but has been met with after interference with the pulley of the superior oblique in frontal sinus operations. The vertical defect is made manifest by placing the head straight, when diplopia is also elicited. Partial myomectomy of the inferior oblique on the side opposite to the direction of the head-tilt corrects the deformity in some cases.

(5) *Vertigo, &c.* Vertigo, leading to nausea, and even vomiting, is due partly to diplopia, partly to false projection. It occurs chiefly when the paralysed muscle is called upon to exert itself. When the gaze is turned from the region of correct to that of false localisation, objects appear to move with increasing velocity in the direction in which the eye is moving. The unpleasant symptoms are counteracted partially by altering the position of the head, or completely by shutting or covering the affected eye.

In paralyses of long standing, false orientation gradually ceases (*vide supra*). Diplopia also tends to disappear or become less troublesome; the patient learns to ignore the impressions derived from the affected eye. Contracture of the antagonists of the paralysed muscle gradually sets in, which has the effect of increasing the primary deviation. Since the retinal image is thus thrown farther to the periphery, where the sensitiveness is less (*vide* p. 66), its suppression is facilitated.

*Investigation of a Case of Ocular Paralysis.* The patient usually seeks advice on account of diplopia. In some cases the nature of the case is obvious immediately from the strabismus or from the manner in which the head is held. In most cases these features are too slight to decide the diagnosis.

(1) The first procedure should be to cover one eye in order to determine whether the diplopia is uniocular or binocular.

(2) Having decided that the diplopia is binocular the patient should fix the surgeon's finger, and the field of fixation of each eye should be carefully investigated (*vide* p. 570). In cases of complete paralysis of one or more muscles it may be possible to make an accurate diagnosis from the observation of the defective movements combined with investigation of the exact positions of the images of the finger in different areas of the field of binocular fixation. In cases of paresis the differentiation of the images is too obscure to permit of the solution of the problem by this means.

(3) In such cases the diplopia must be investigated by more delicate tests. The patient is taken into a dark room. A red glass is placed before one eye in order to distinguish its image. A bar of light through a stenopœic slit in a hand-torch is then moved about in the field of binocular fixation at a distance of at least four feet from the patient, the patient's head being kept stationary. The positions of the images are accurately recorded upon a chart with nine squares marked upon it (Fig. 304). The examination may be carried out by the surgeon turning the patient's head in various directions while the light is kept stationary. The following data are derived from this examination :—

(*a*) The areas of single vision and diplopia ;

(*b*) The distance between the two images in the areas of diplopia ;

(*c*) Whether the images are on the same level or not ;

(*d*) Whether one image is inclined or both are erect ;

(*e*) Whether the diplopia is homonymous or crossed.

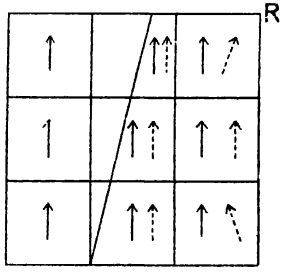



FIG. 304.—Diplopia chart for the right external rectus. The oblique line through the chart shows the limit of the fields of single vision and of diplopia. The dotted arrows show the positions of the false image in different parts of the field of diplopia.

These data, if concordant, are sufficient to diagnose the paralysis. The false image is determined by the direction in which the images are most separated from each other. This is the direction of the normal action of the paralysed muscle. The false image can often be recognised by being the fainter of the two or by being tilted ; by covering one eye it can be shown to which eye this image belongs.

It must be remembered that these tests are purely subjective. In many cases the patients are stupid or their intelligence is obscured by intracranial disease ; or contracture of the antagonistic muscles may have set in. Consequently the answers are not infrequently discordant, and accurate diagnosis may be extremely difficult or impossible. There are two not infrequent causes of ambiguity. The paresis may unmask a latent squint (*vide* p. 588), or the patient may fix with the paralysed eye, especially if this eye has the greater acuity of vision.

The nature of the diplopia and the position of the images in each of the nine areas of the field of fixation for paralysis of each individual muscle should be worked out by the student. In performing this exercise he should rely upon his knowledge of the anatomy of the muscles and their consequent action in each position of the eye. Considerable ingenuity has been used to devise mnemonics for determining the position

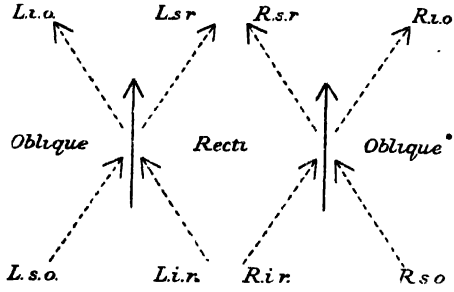



FIG. 305.—Werner's mnemonic for paralyses of elevator and depressor muscles. Note that the right muscles are to the right, the left to the left; recti in centre, obliques outside, superior recti above, superior obliques below. The diagram illustrates the normal actions of the muscles and the type of diplopia caused. For example:—*Actions:* The recti adduct, the obliques abduct; the superior muscles (*R.s.r.*, *L.s.r.*, *R.s.o*, *L.s.o.*) produce inward torsion, the inferior muscles outward torsion; the movement follows the direction of the arrow, *e.g.*, *R.s.r.* moves the eye inwards and upwards and causes inward torsion. *Diplopia:* The false image (broken arrows) is displaced in the direction of action of the muscle; therefore, for muscles in the upper half (*L.i.o.*, *L.s.r.*, *R.s.r.*, *R.i.o.*) the diplopia occurs on upward movement of the eyes, and the false image is higher than the true. The diagram also shows whether the diplopia is homonymous or crossed. If the patient fixes with the paralysed eye the figure must be rotated so that the false image becomes vertical.

of the false image. One of the most satisfactory is shown in the accompanying diagram (Fig. 305). It may be pointed out that all the signs, with the exception of the deviation of the eye, viz., defective movement, false projection, increase of diplopia, secondary deviation, and position of the head, are towards the side of the paralysed muscle.

*Varieties of Ocular Paralysis.* If one muscle alone is affected it is generally the external rectus or the superior oblique, since each of these is supplied by an independent nerve.

Affection of several muscles simultaneously is usually due to paralysis of the third nerve. All the extrinsic and intrinsic muscles of one or both eyes may be paralysed—*ophthalmoplegia*

*totalis* (*vide* p. 601). If only the extrinsic muscles are affected the condition is called *ophthalmoplegia externa;* if only the intrinsic (sphincter pupillæ and ciliary muscle) *ophthalmoplegia interna.*

*Conjugate paralysis* is the term applied to abolition of certain synkineses. Thus ability to look up, to the right or left, or down may be lost. Inability to converge may also occur. Such defects might be conveniently termed *symparalyses.*

*Paralysis of the External Rectus.* There is limitation of movement outwards, and the face is turned towards the paralysed side. Diplopia occurs on looking to the paralysed side. It is homonymous; the images are on the same level, and erect, becoming more separated on looking more towards

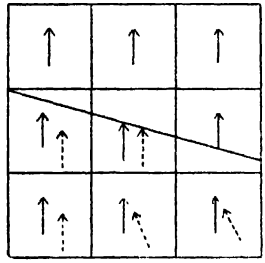


FIG. 306.—Diplopia chart for the right superior oblique.

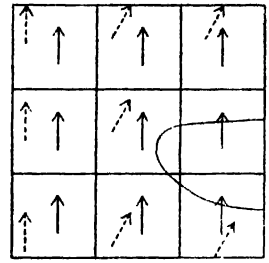


FIG. 307.—Diplopia chart for the right third nerve. The area enclosed by the curved line is the area of single vision.

the paralysed side. The false image is slightly tilted on looking up or down as well as towards the paralysed side (Fig. 304).

*Paralysis of the Superior Oblique.* There is limitation of movement downwards and towards the paralysed side; the face is turned downwards and towards the *sound* side. Diplopia occurs on looking down (Fig. 306). It is homonymous; the false image is lower and its upper end is tilted towards the true image. The distance between the images and the inclination of the false image increase on looking down and towards the paralysed side. The patient has great difficulty in going downstairs, and vertigo is usually a particularly prominent symptom.

*Paralysis of the Third Nerve.* In complete paralysis of the third nerve there is ptosis, which prevents diplopia. On raising the lid with the finger the eye is seen to be deflected outwards and somewhat downwards, owing to the tone of the

two unparalysed muscles. The pupil is semi-dilated and immobile, and accommodation is paralysed. There is a slight degree of proptosis, owing to loss of tone of the paralysed muscles. There is limitation of movement upwards and inwards, to a less degree downwards. With the lid raised there is diplopia, which is crossed, the false image being higher, with its upper end tilted towards the paralysed side (Fig. 307).

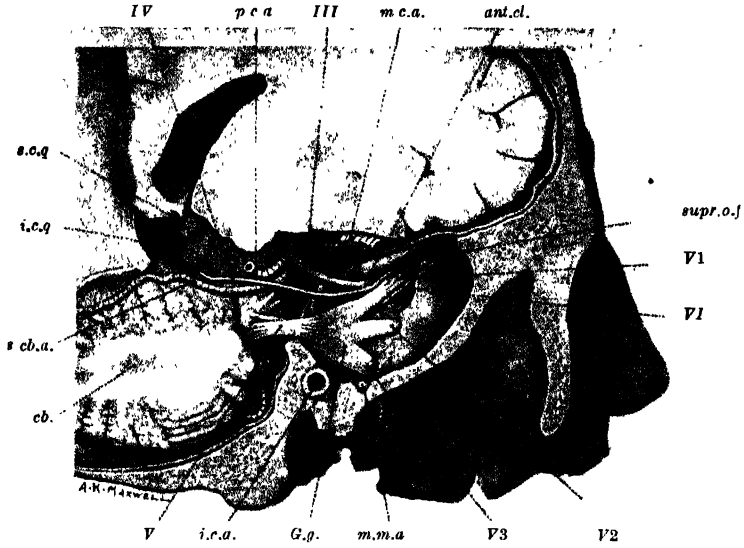



FIG. 308.—Third, Fourth, Fifth and Sixth Nerves. *G.g.*, Gasserian ganglion; *i.c.a.*, internal carotid artery; *m.m.a.*, middle meningeal artery; *p.c.a.*, posterior cerebral artery; *m.c.a.*, middle cerebral artery; *supr.o.f.*, superior orbital fissure; *s.c.q.*, superior corpus quadrigeminum; *i.c.q.*, inferior corpus quadrigeminum; *s.cb.a.*, superior cerebellar artery; *cb.*, cerebellum. (Eugene Wolff. "Anatomy of the Eye and Orbit." Lewis, London.)

Paralysis of the third nerve is often incomplete, and individual muscles may occasionally be affected alone.

*Ætiology.* Paralysis of ocular muscles may result from a lesion situated in any part of the nerve tracts from the cerebral cortex to the muscles. The site may therefore be intracranial or intraorbital. Cortical lesions usually cause loss of synergic movements, *e.g.*, conjugate deviations, but simple ptosis may be due to such a lesion. The diagnosis of nuclear and peripheral lesions depends largely upon knowledge of the

anatomical relations of the nuclei and nerves. It is beyond the scope of this work to treat the subject exhaustively here. It may be mentioned that paralysis of the external rectus, sometimes bilateral, is common in babies. It may be due to the use of forceps during delivery, the sixth nerve being most exposed to pressure, or to maldevelopment of the nucleus. In the latter case there is loss of conjugate deviation to the same

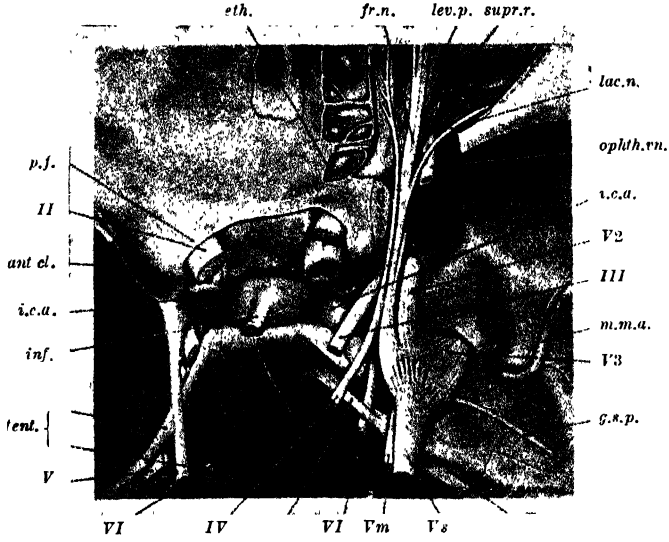



FIG. 309.—Third, Fourth, Fifth and Sixth Nerves. *eth.*, ethmoidal cell; *fr.n.*, frontal nerve; *lev.p.*, levator palpebræ; *supr.r.*, superior rectus; *lac.n.*, lacrymal nerve; *opth.vn.*, ophthalmic vein; *i.c.a.*, internal carotid artery; *m.m.a.*, middle meningeal artery; *g.s.p.*, great superficial petrosal nerve; *tent.*, tentorium; *inf.* infundibulum; *ant.cl.*, anterior clinoid process, *p.f.*, processus falciformis. (Eugene Wolff. "Anatomy of Eye and Orbit." Lewis, London.)

side (*vide* p. 546), and the seventh nerve is normal. In acquired sixth nuclear paralysis in adults the seventh nerve is usually implicated (*vide* p. 546). In congenital paralysis of the external rectus contracture of the antagonists does not occur. Other nuclei may be maldeveloped, or they may be picked out by the lesions of syphilis, tabes, disseminated sclerosis, or polioencephalitis.

The commonest cause of ocular paralyses is syphilis, which may affect the nerves at their origin or in any part of their

course (see Section VI.). Syphilitic paralysis is usually a late manifestation. The third nerve is affected most frequently, but not necessarily equally in all its branches. Tabes is responsible for a large proportion of the cases (*vide* p. 594).

Ocular paralyses of intracranial origin may be due to affec-

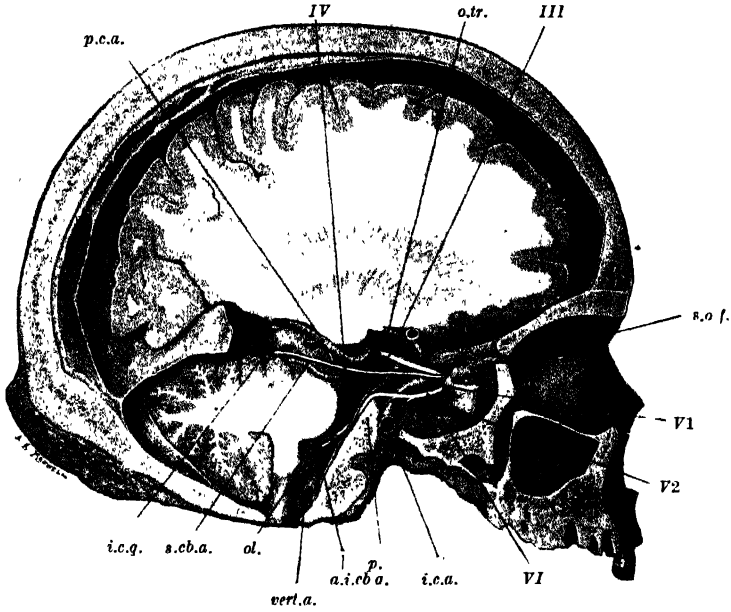



FIG. 310.—Course of Sixth Nerve. *p.c.a.*, posterior cerebral artery; *o.tr.*, optic tract; *s.o.f*, superior orbital fissure; *i.c.a.*, internal carotid artery; *p.*, petrous portion of temporal bone; *a.i.cb.a.*, anterior inferior cerebellar artery; *vert.a.*, vertebral artery; *ol.*, olive; *s.cb.a.*, superior cerebellar artery; *i.c.q.*, inferior corpus quadrigeminum. (Eugene Wolff. "Anatomy of the Eye and Orbit." Lewis, London.)

tions of the blood vessels—hæmorrhage, thrombosis, &c.—or to external pressure—tumours, bloodclots, periostitis, &c.

Other causes are diphtheria, diabetes and other toxic conditions, injury, &c. Paralysis of both extrinsic and intrinsic ocular muscles is a common and early feature in encephalitis lethargica. Paralysis, especially of the external rectus, sometimes follows spinal anæsthesia with stovain; the onset is rapid, and recovery usually takes many weeks. Ophthalmoplegic migraine is a rare cause (*vide* p. 416). Involvement of the cranial nerves near the cavernous sinus,

especially the sixth, associated with trigeminal neuralgia, is an early symptom of malignant nasopharyngeal tumours (*vide* p. 615).

Paralysis of the external recti is common in cases of intracranial tumours with high intracranial pressure, and generally has no localising value. It may be due to traction on the nerves as they bend over the apex of the petrous portion of the temporal bone (Wolff, Figs. 310, 311), or to pressure by the anterior inferior cerebellar and internal auditory arteries, which cross them at right angles and often lie ventral to them (Fig. 325)

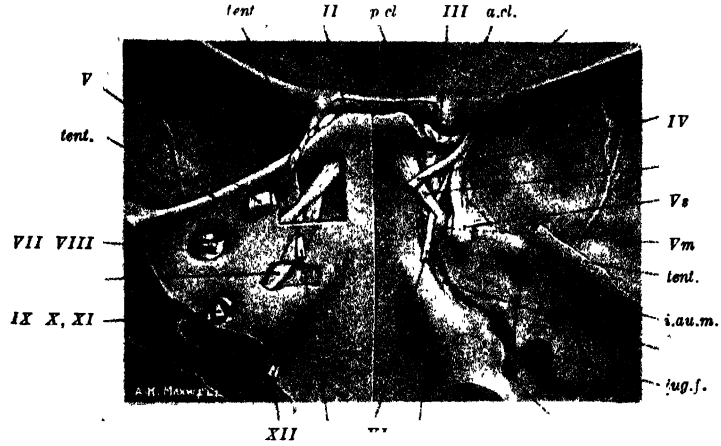



FIG. 311.—Course of Sixth Nerve. *tent.*, tentorium; *a.cl.*, *p.cl.*, anterior and posterior clinoid processes; *i.c.a.*, internal carotid artery; *i.au.m.*, internal auditory meatus; *jug.f.*, jugular foramen. (Eugene Wolff. "Anatomy of the Eye and Orbit." Lewis, London.)

—the nerves are strangulated between the vessels and the œdematous and swollen pons (Cushing). This may also account for the spinal anæsthetic cases, and for ophthalmoplegic migraine, the third nerve passing between the superior cerebellar and the posterior cerebral arteries.

Injury to and disease of the orbit may affect the nerves of muscles in this situation by rupture, pressure, inflammation, &c.

The prognosis varies with the cause. Ocular paralyses are so often early signs of grave nervous disease that the prognosis should always be guarded. Those due to peripheral disease, dependent upon syphilis or "rheumatism," may speedily recover. Long-standing cases rarely recover.

*Treatment.* Syphilitic cases should be treated by mercury and increasing doses of iodides, and this treatment should be applied to all doubtful cases. N.A.B. should be tried in intractable cases. Some cases improve on salicylates, or colchicum and iodides. Diaphoresis may be used. The constant current is chiefly useful in keeping up the metabolism of the muscles until innervation is re-established. It probably has little therapeutic influence upon the paralysed nerve.

Occasionally symptomatic treatment affords relief to the patient. The diplopia may sometimes be relieved by suitable prisms, but this treatment is rarely of much use owing to the variation in the amount of the deviation in different positions of the eyes. Occasionally good is done by exercising the weak muscle with strong prisms (*vide* p. 593). In old cases an operation may be indicated, usually tenotomy of the antagonist with advancement of the paralysed muscle, thus putting the affected muscle under better mechanical conditions. It is only suitable for paretic, not paralytic, cases, and should never be adopted until all other means have failed. It is therefore seldom indicated.

If diplopia is very troublesome and cannot be relieved by the means suggested, spectacles should be ordered with a ground glass in front of the affected eye.

## Kinetic Strabismus

Aberrant forms of strabismus occur as the result of irritative intracranial lesions, and are due, not to paralysis, but to irregular action or over-action of certain muscles, caused by unequal stimulation of the nerve centres or nerves. Such squints are common in meningitis and lesions of the mid-brain or cerebellum, such as tumours (glioma, tubercle, gumma, &c.). The occurrence of the squint only during epileptiform fits or its irregularity of type may render the diagnosis from paralytic squint easy, especially when there are other prominent symptoms of cerebral irritation. In other cases, especially in the early stages of the disease, the diagnosis from paralytic or concomitant squint may be extremely difficult.

## Synkineses

The extrinsic muscles take part in many normal and pathological synkineses. When the eyes look up the levatores palpebrarum raise the lids and in extreme upward movements the frontales also contract. In congenital ptosis (*q.v.*) upward move-

ment of the eyes is often defective. On looking down the lid follows the globe. In exophthalmic goître the lid follows tardily or not at all (von Graefe's sign); in total facial paralysis the lid follows the globe on looking down, though the eye cannot be closed voluntarily. On closing the eyes, as in sleep, the eyes generally turn upwards and outwards. The same movement of the eyes occurs on attempted closure in total facial paralysis. On the other hand, the eye sometimes closes in total seventh nerve paralysis on synergic activity of other facial muscles, as in laughing. The so-called "jaw-winking" synkinesis is particularly striking. In these rare cases one levator palpebræ is thrown into activity during eating, and sometimes on reading aloud. The lid movement is usually specially associated with lateral movements of the jaw, due to action of the pterygoid muscles, which are innervated by the fifth nerve. In most cases, but not all, there is slight ptosis of the affected lid, and in cases with congenital ptosis the synkinesis occurs on sucking. Allied to the jaw-winking cases are others in which spasmodic lid movements occur on lateral deviation of the eyes. The convergence pupillary synkinesis has already been mentioned: to it may be added the contraction of the pupil on forced closure of the lids. In rare cases spontaneous rhythmical variations in the size of the pupil are accompanied by ocular or lid movements. They are usually associated with congenital or early infantile paresis of the third nerve. The pupil contracts rapidly to about 2 mm. diameter, then after 5—10 seconds dilates slowly to 6—7 mm., contracting again after 15—20 seconds. Contraction is hastened by action of the internal rectus, dilatation by action of the external rectus. The movements are accompanied by spasm and relaxation of the ciliary muscle.

### Nystagmus

Nystagmus (*νυσταζειν*, to nod) is the term applied to rapid oscillatory movements of the eyes, independent of the normal movements, which are not affected. The oscillations are involuntary, though in rare cases normal persons can imitate them. They are usually lateral, but vertical, rotatory, and mixed rotatory and lateral or vertical nystagmus are not uncommon. The condition is almost always bilateral, though the movements may be much more marked in one eye than the other. In such cases it may be necessary to examine the eye very carefully with the ophthalmoscope (corneal reflex, retinal vessels, &c.) before the presence of nystagmus can be demonstrated. Unilateral nystagmus does occur, but it is probable that many of the cases described are really bilateral.

Nystagmoid jerks, *i.e.*, larger rhythmic jerking movements,

most pronounced at the extreme limits of the normal movements of the eyes, should be distinguished from true nystagmus. They are not uncommon in normal people under certain conditions—fatigue, railway travelling, &c. The fundamental cause is probably quite different from that of true nystagmus, though both may occur together.

Nystagmus may be congenital or early infantile, or it may be acquired. These two groups of cases should also be carefully distinguished on account of their different pathological foundation. Congenital and early infantile nystagmus, *i.e.*, nystagmus dating from birth or within a few weeks of birth, occurs in congenitally malformed eyes, in albinism, and in eyes with congenital or early developed opacities of the media, *e.g.*, leucoma or anterior polar cataract due to ophthalmia neonatorum (*q.v.*), macular changes, &c. The cause in these cases is inability to develop normal fixation. Fixation is developed during the first few weeks of life, the eyes being moved aimlessly and independently before it is acquired. Any cause seriously diminishing the acuity of macular vision occurring at this period is liable to give rise to nystagmus; if the eye is blind, nystagmus is not developed. Nystagmus is present in most cases of total colour blindness (*q.v.*), in which vision is carried out by the rods alone, and there is therefore a central scotoma. In some congenital cases it is impossible to discover any cause. In a few such cases ancestors or relations have been albinos.

Nystagmus may be acquired in infancy after the period at which fixation is developed. This form occurs in *spasmus nutans*, in which it is associated with nodding movements of the head. It occurs in the first year of life. The nodding of the head may be antero-posterior (affirmation), lateral (negation), or rotatory. It develops some weeks before the nystagmus, ceases during sleep, and disappears before the nystagmus. The nystagmus is very fine and rapid, and may be vertical, rotatory, or lateral. It is generally more marked in one eye. The whole symptom-complex disappears in time—one of the few cases in which nystagmus disappears spontaneously. The nystagmus may disappear in one eye before the other; such cases may be mistaken for true unilateral nystagmus. In rare cases head nodding with nystagmus is congenital and hereditary, and in these cases persists throughout life (Hancock).

Nystagmus in adults occurs in disseminated sclerosis, disease of the cerebellum and vestibular tracts, and of the semicircular canals (*e.g.*, occasionally on syringing the ears), Friedreich's ataxia, &c. In disseminated sclerosis the movements are

generally horizontal and are elicited in the early stages only in extreme lateral positions of the eyes. Cerebellar irritative lesions cause coarse nystagmus towards the side of the lesion and fine nystagmus to the opposite side. Some of these cases show analogy with hippus (*vide* p. 61), and like it are probably dependent upon the rhythmic activity of nerve centres. Nystagmus may also occur in adults as an "occupation neurosis," the commonest form being coal-miners' nystagmus (*vide infra*).

In congenital and early infantile nystagmus the patient is wholly unconscious of the movements, since objects do not appear to move. Vision is usually defective in spite of correction of errors of refraction which generally accompany the defect. In some cases of acquired nystagmus in adults objects appear to move.

The prognosis is good in spasmus nutans, and in miners' nystagmus if the occupation is changed, though recovery is slow. In all other cases it is bad, though it tends to diminish with advancing years. Treatment is therefore palliative, consisting in correction of refraction, wearing smoked glasses in albinism, and treating any disease which may be present.

**Labyrinthine Nystagmus** occurs in disease of the internal ear in which the semicircular canals are involved, and can be produced in normal subjects by rotation in a specially designed chair or by passing a galvanic current through the head. The nystagmus is rhythmic, with a rapid and a slow component, is bilateral, and horizontal or rotatory, but varies according to the semicircular canal stimulated. Either pair of semicircular canals can be stimulated by rotation with the head in a suitable position. Destruction of one labyrinth causes rhythmic nystagmus towards the opposite side, which ceases if the other labyrinth is destroyed.

**Miners' Nystagmus** occurs chiefly in those who have worked long at the coal face. The patient complains of defective vision which is worse at night, headache, giddiness, photophobia, dancing of lights and movements of objects. The nystagmus is essentially rotatory and very rapid; in latent cases it is elicited by fixing the head and making the patient look up. In severe cases the lids are nearly closed and the head is held backwards; there is tremor of the head and eyebrows. The disease is six times as common in pits which use safety-lamps as compared with those that use naked lights, and varies inversely with the illumination (Llewellyn). Continual looking upward at work is only a contributory cause, other such causes being ill-health, accidents, errors of refraction, subnormal pigmentation, &c.

Scotopia, or vision in a dull light, is carried out almost entirely by the rods. Under these circumstances visual acuity is greatest 10°—15° outside the fovea, and there is a physiological central scotoma. There is great difficulty in keeping up fixation, and the evidence is strongly in favour of the view that low illumination is the essential ætiological factor in miners' nystagmus. Improvement in miners' lamps and in the lighting of mines would probably eliminate the disease, which is a cause of enormous economic loss in compensations, &c. There is, however, a large psychoneurotic factor in all cases.

# CHAPTER XXVIII

## Concomitant Strabismus Heterophoria Congenital Defects

### Concomitant Strabismus

In concomitant strabismus the visual axes, though abnormally directed, retain their abnormal relation to each other in all movements of the eyes. It differs therefore in this respect from paralytic strabismus, in which the relationship of the visual axes to each other changes with every movement of the eyes in the direction of action of the paralysed muscle or in the resultant direction of action of the paralysed muscles when more than one is affected. The secondary deviation in concomitant squint is equal to the primary deviation, a fact which demonstrates the absence of paralysis (Figs. 312, 313). The deviation of the visual axes may be convergent or divergent, the former being the more common.

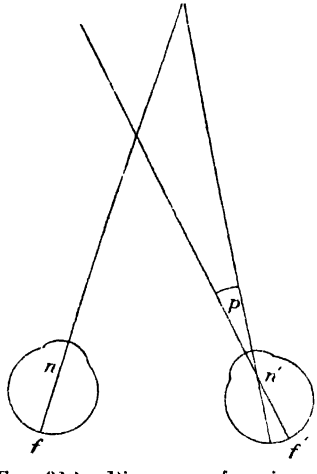


Fig. 312.—Diagram of primary deviation in concomitant convergent strabismus.

In every case in which the direction of the visual axes is aberrant paralytic strabismus must first be eliminated by testing the movements of the eyes in all directions with the finger. If they are found to be normal and there is no complaint of diplopia, it may be concluded that there is no paralysis. It does not follow that there is concomitant squint. The convergence or divergence of the axes may be only apparent. A marked appearance of convergent squint is sometimes seen in myopic eyes, of divergent squint in hypermetropic eyes. It will be observed later that true concomitant

convergent squint is most commonly associated with hypermetropia, divergent with myopia, *i.e.*, the opposite of apparent strabismus.

*Apparent strabismus* is due to the fact that the visual axis of the eye is very rarely coincident with the optic axis (Fig. 314). The optic axis, *i.e.*, the axis upon which the cornea and lens are centred, passes through the centre of rotation of the eye and approximately through the centre of the pupil. The visual axis passes through the nodal point and the fovea centralis, thus crossing the optic axis and making a small angle with it. This angle is very nearly equal to an angle which is called the angle gamma; it is commonly spoken of clinically as the angle $\gamma$. In the emmetropic eye the angle $\gamma$ is said to be positive, *i.e.*, the optic axis cuts the retina internal to the fovea centralis. In hypermetropic eyes the angle $\gamma$ is also positive but greater than in emmetropia. In myopia the angle $\gamma$ is absent or negative, *i.e.*, the visual axis and the optic axis coincide or the latter cuts the retina external to the fovea centralis.

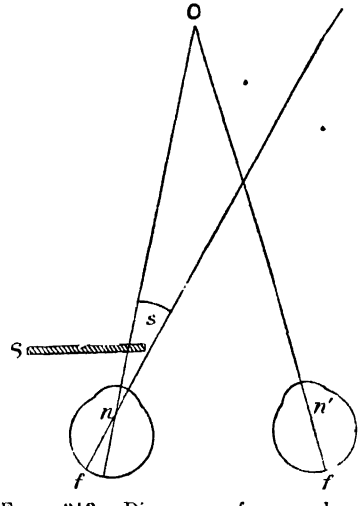



FIG. 313.—Diagram of secondary deviation in concomitant convergent strabismus.

Now, neither of these lines can be seen, and the direction of the line of vision is judged by the position of the pupil. Hence the greater the size of a positive angle $\gamma$ the more the eye will appear to look outwards. If the angle $\gamma$ is negative the eye will appear to look inwards. Therefore in high hypermetropia there will be an apparent divergent squint, in high myopia an apparent convergent squint. The latter is the more striking because the emmetropic eye usually has a positive angle $\gamma$ of 5°, thus producing an apparent divergence of 10°, which, however, we are accustomed to regard as the normal position of the eyes.

Having decided that the case is not one of paralytic strabismus, it is necessary next to show that it is real, not merely apparent. This is easily done as follows: The patient is told

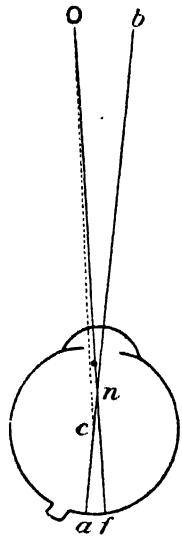



FIG. 314.—Apparent strabismus. *a b*, optic axis upon which the refractive surfaces are centred; *f*, fovea centralis; *n*, nodal point; *c*, centre of rotation; O, point of fixation; O *c*, line of fixation; O *n f*, line of vision; O *c b*, angle $\gamma$. It is practically equal to O *n b*, which can be measured. In actual practice the guide to *a b* is taken from the centre of the pupil; *a b* does not usually pass accurately through the centre of the pupil, so that the result is always only approximate. The angle $\gamma$ is to the nasal side in hypermetropia and emmetropia.

to fix the surgeon's index finger, which is held up at least two feet from the eyes. If it is held closer, as is too often done, normal convergence will vitiate the result. The surgeon's left hand or a screen is held in front of the patient's right eye; the left eye will now be accurately fixing the finger. The screen is then moved so as to cover the left eye, fixation being now taken up by the right eye. If the right eye moves inwards or outwards at the moment when it takes up fixation there is a true squint; if it remains absolutely motionless the squint is apparent only.

Having now eliminated both paralytic and apparent strabismus, it is almost certain that the case is one of true concomitant squint (see, however, p. 584). In concomitant squint one eye maintains fixation while the other is adducted or abducted. If in the test just described the fixing eye is covered by the screen, the deviating eye generally moves outwards or inwards through an angle equivalent to the angle of the deviation in order to take up fixation. At the same time the eye behind the screen moves inwards or outwards through exactly the same angle (secondary deviation). If now the screen is removed it will generally be found that fixation immediately reverts to the first eye. Such a case is usually termed one of *unilateral strabismus*. In some cases fixation is still retained by the second eye. If it is so retained for a considerable period, *e.g.*, while the patient blinks several times, the squint is said to be *alternating*. Inquiry will then generally elicit the fact that the patient sometimes squints with one eye, sometimes with the other: indeed, this may be noticed while watching the patient. Usually an object towards the right in the field of vision will

be fixed with the right eye, in the left of the field by the left eye. Occasionally patients with alternating strabismus can fix with either eye voluntarily, but usually they are unconscious which eye is fixing. Concomitant squint may be *constant*, or occur only at intervals—*periodic*.

It has been mentioned that when the fixing eye is covered with the screen the deviating eye usually moves so as to take up fixation. In unilateral squints of long standing this eye may remain motionless and can only be moved into the primary position by moving the finger, a condition which is called *eccentric fixation* (*vide* p. 574). Since it occurs only with marked deviation of long standing there is generally no difficulty in distinguishing it from apparent squint.

In performing the preliminary test to eliminate paralytic strabismus it will often be found that in true concomitant squint with considerable deviation the eyes do not move as much as usual in the direction opposite to that of the deviation. Thus, in convergent squint it may be very difficult to get the eyes to move outwards as much as normal, *i.e.*, so that the margin of the cornea lies under the external canthus. Similarly in divergent squint it may be very difficult to get the eyes to move inwards as much as normal, *i.e.*, so that the margin of the cornea is well covered by the internal canthus. This defective movement is commonly attributed to "insufficiency" of the external or internal recti respectively. In convergent strabismus it is probably due, not to any defect in the external rectus or its innervation, but to the fact that, fixation being dependent upon one eye, there is little stimulus to outward movement as soon as the point of fixation has passed beyond the field of fixation of this eye, *i.e.*, as soon as the nose cuts off vision of the finger. In many such cases the eye will move out completely if the finger is moved rapidly. In very young children it is better to turn the child's head in the opposite direction, in which case curiosity stimulates the child to keep up fixation. In divergent strabismus defective inward movement is sometimes due to mechanical causes, viz., the size of the myopic eye (*vide* p. 584).

It has already been mentioned that in concomitant strabismus there is no diplopia. It may be present in the earliest stages, but is invariably absent in the later. This is due to psychological suppression of the image of the squinting eye. In most cases suppression is aided by actual defect, usually ametropia, in this eye, but such is not the complete explanation, since suppression of the image of the squinting eye is also the

rule in alternating squint, in which both eyes are frequently quite normal or have the same degree of ametropia. Suppression is doubtless aided in all cases by the peripheral situation of the image in the squinting eye, but there is no doubt that the seat of suppression is really in the brain; that is, in the interpretation of the stimuli reaching the brain from the eyes those derived from the squinting eye are unwittingly neglected. The ability to exclude the impulses derived from the squinting eye is an important point in attempting to arrive at the rationale of concomitant strabismus. It follows from this fact that people with convergent squint have only uniocular vision, or, at most, very imperfect binocular vision.

Except in alternating strabismus the vision of the squinting eye is nearly always defective, which is partly due in most cases to errors of refraction. In convergent strabismus the eyes are nearly always hypermetropic, with or without astigmatism, and often the squinting eye has greater ametropia. There can be little doubt that some such inherent defect may determine which eye will deviate, though it is probably never the fundamental cause of the squint.

The vision in the squinting eye is often defective beyond any explanation derived from objective defects—ametropia, &c. In some such cases there is reason to believe that the defective vision dates from birth—*congenital amblyopia*. In all unilateral squints of long standing, very defective vision in the squinting eye is the rule. It is commonly attributed to the prolonged suppression of the images derived from this eye, and is hence called *amblyopia ex anopsiâ*. This explanation is not altogether satisfactory, since cases are well known in which vision has been excluded for many years by congenital cataract, yet is quite good after successful operation. The vision in the amblyopic eye is often reduced to 6/60, and may be reduced to counting fingers. Cases of recovery of sight after loss of the fixing eye have been recorded, but unfortunately this result certainly does not invariably follow. The visual acuity may be greater in the false position than when the retinal image falls upon the fovea (eccentric fixation with "false macula," abnormal retinal correspondence). This results in "false projection," or all power of fixation may be lost by the amblyopic eye.

It has already been mentioned that the gross movements of the eyes are nearly or quite perfect in concomitant strabismus. This applies equally to dynamic convergence and accommodation. The eyes start in an abnormal position, and normal

movements are superposed. Thus, in fixing a new object, the normal amount of dynamic convergence is superposed upon the abnormal static convergence or divergence.

Concomitant strabismus always commences in childhood, generally in infancy. It may become manifest after a fright, an attack of whooping cough, measles or other debilitating illness, and is often popularly attributed to some such cause. It must be carefully distinguished from the squinting which normally occurs during the first few weeks of life, before fixation is developed (*vide* p. 567); this is not concomitant. Many important factors in the ætiology of concomitant strabismus are known, and a proper appreciation of them is essential to rational treatment. No theory of the fundamental causation which has yet been advanced satisfactorily explains the condition.

Attention was early drawn by Donders to the common association of convergent strabismus with hypermetropia. He explained the relationship by the fact of the normal association of convergence and accommodation (*vide* p. 549). Hypermetropes have to exercise an effort of accommodation to see distant objects, still more to see near objects. The effort of accommodation is associated in the normal person with a corresponding effort of convergence. If this rule be supposed to hold good for hypermetropes, the strong accommodation may be regarded as inciting an effort of convergence which is excessive for the actual point of fixation. The hypermetrope is therefore in a dilemma. He must either converge accurately for the object, in which case he will not accommodate sufficiently to see it clearly: or he must accommodate accurately for it, in which case he will converge too much. This will cause homonymous diplopia, unless he is able to suppress the image of one eye, which is exactly what the patient with convergent strabismus does. Regarding divergence as negative convergence, the association of divergent strabismus with myopia is explained by the same theory.

There is no question that this factor is one of great importance, as is shown by the cure of some cases of squint by suitable correcting glasses. It is not, however, the fundamental cause of squint, for if it were (1) all uncorrected hypermetropes would have to squint; (2) there would be ametropia in all cases of concomitant strabismus. The latter corollary is found to be false, for convergent strabismus, other than apparent strabismus, is occasionally found to be associated with myopia, and in alternating strabismus there is often little or no ametropia.

In spite of these facts the great importance of the association between accommodation and fixation must be strongly insisted upon. Convergent strabismus most frequently develops between the ages of two and six, *i.e.*, just at the period when the fixation of near objects throws a strain upon accommodation. It is often periodic at this stage, and noticed only when near objects are looked at. Moreover, there is an undoubted tendency for the deviation in all cases of convergent strabismus to diminish with age, *i.e.*, with the diminution of accommodation. The relative infrequency of convergent squint in adults, compared with its frequency in children, cannot be explained solely by the fact that many cases undergo successful treatment. It may be remarked that the amblyopia persists in the formerly squinting eye; hence in every case in which satisfactory objective evidence of the cause of defective vision in one eye cannot be discovered, the patient should be asked if he ever squinted.

As already mentioned, greater ametropia in one eye, opacities in the refracting media, intraocular disease, and so on, are never the fundamental cause of strabismus, though they may determine the particular eye which loses fixation. This is especially the case when concomitant squint is preceded by latent squint (*q.v.*). It is easy to understand that when there is disturbance of muscular equilibrium which can only be overcome by special effort, any slight defect in one eye may determine the development of a manifest squint.

The application of Donders' theory to divergent strabismus associated with myopia has been mentioned. Here, since near objects are seen with little or no accommodation, the impulse to convergence is too weak. Since infants are rarely myopic, this form of divergent squint does not develop in early childhood. There are other factors besides deficient accommodation which tend to produce divergence in myopia. One is the mechanical condition of the myopic eyeball, which, being abnormally large and long, adapts itself to the axis of the orbit. Further, the internal recti act under mechanical disadvantage from the same cause. Moreover, in very high myopia the far point of the eye is so close to it that it is impossible for convergence to be effectual—it becomes impossible to see the object with both eyes at the same time. The better eye is then used and the other is allowed to take up the position of rest, which is usually one of divergence. Such a strabismus may remain periodic for near work only for many years; in other cases it becomes constant.

Spontaneous cure rarely if ever occurs in divergent strabismus, which tends to increase with age.

The deviation in convergent squint is not always quite horizontal: in many cases the eye deviates upwards as well as inwards. In some cases the deviation is still more unusual, and the movements of the eye are quite abnormal. In most of these cases there is a congenital mal-insertion or defective development of one or more of the extrinsic muscles and the squint dates from birth (*vide* p. 588).

Concomitant strabismus has proved a fertile field for conjecture. There are several points bearing upon the subject which are obscure, such as the development of fixation and of binocular vision, the occurrence of congenital amblyopia, and of amblyopia ex anopsia, and so on. Many theories relating to these points have been stated and restated so frequently that they are accepted as facts. In reality, many are not susceptible of demonstration, and none have been proved. The theorist on strabismus accepts the "facts" which fit his theory, and rejects the remainder.

The prevailing theory at the present time is that strabismus is due to defect of the fusion faculty, or the capacity of combining psychologically the impressions derived from the two eyes. Some authors go so far as to hypothesize a "fusion centre" in the brain. No one will deny that the fusion of the images derived from the two eyes in binocular vision is a mental though unwitting act, and that it has a physical basis. This physical basis is a set of accurately co-ordinated nerve impulses. Binocular vision, then, depends upon the accuracy of co-ordination of these impulses, and this is a function of the nerve-complex as a whole, not of any particular "centre." It is indeed possible that the strabismus is caused by inco-ordination of the afferent impulses upon which binocular vision depends, though this advances the true ætiology of the disease but little. It is just as probable that the inco-ordination or deficiency of the afferent impulses is a result of the strabismus, which is itself due to some other cause; or both may be due to a common cause.

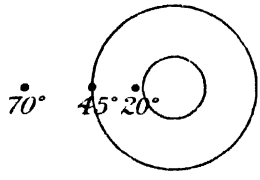



FIG. 315.—Diagram of the position of the corneal reflex as a guide to the angle of the squint.

In every case of concomitant strabismus the angle of the deviation should be measured, so that the mode of treatment

may be determined and its effects accurately gauged. A rough indication of the angle of the squint can be obtained from the position of the corneal reflex when light is thrown into the eye with the ophthalmoscopic mirror (Fig. 315). The light is thrown in from a distance of about two feet, and the patient is told to look at the mirror; an infant does this reflexly. In the fixing eye the corneal reflex will be in the centre of the pupil, or slightly to the inner side if there is a large angle $\gamma$, to the outer side if there is a negative angle $\gamma$. The light is then turned on to the squinting eye. If the reflex is about half-way between the centre of the pupil and the corneal margin, there is a deviation of about 20°; if it is at the corneal margin, about 45°. This test is only a rough one.

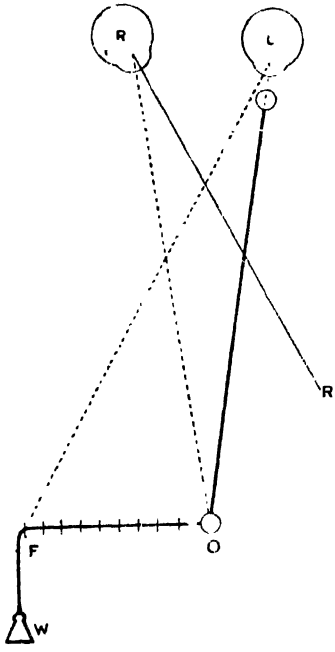



FIG. 316.—Diagram of Priestley Smith's tape for measuring the angle of squint. R, right eye with internal squint; L, left eye; O, ophthalmoscope; O F, graduated tape; F, fixation point (observer's finger); W, weight. The angle measured is O L F, which is equal to O R R′, the angle of the squint.

The most accurate test for general application is by the use of a major amblyoscope by which the squint can be measured both objectively by observing the corneal reflex and subjectively by using each eye alternately for fixation. In the case of children attention can be maintained by the use of attractive pictures.

An alternative test for children is with Priestley Smith's tape (Fig. 316). It consists of a tape 1 metre or 60 cm. long, with a ring at each end. To one ring a second tape is attached, having a tangent scale upon it. The graduations are, of course, different, according to the length of the first tape. At the other end of the tangent tape is a small weight. The measurement is carried out in the dark room. The free ring is held by the patient or an assistant on the cheek immediately below the fixing eye. The surgeon passes one finger of the hand, which also holds

his ophthalmoscope, through the other ring, and keeps the tape taut. With his disengaged hand he holds the tangent tape at right angles to the distance tape, at the same time holding up the index finger as an object of fixation. The light from the ophthalmoscope is thrown into the squinting eye, and the patient is told to look at the fixation finger. This is moved along the tangent tape until the corneal reflex is in the centre of the pupil. The angle of the squint is then read off on the tape. The direction in which the tangent tape is held, whether to the surgeon's right or left, depends, of course, upon the side of the squinting eye and the nature of the squint, whether convergent or divergent. This test is the best yet devised for small children.

The angle of deviation can also be measured with the perimeter (Fig. 317). The squinting eye is placed at the centre of the arc, and the patient fixes an object six metres away, situated slightly over the position of the ordinary fixation spot of the perimeter. The arc of the perimeter is turned to the horizontal position on the side towards which the squinting eye is directed. The surgeon passes a light (as from an electric ophthalmoscope or torch) along the arc until the corneal reflex is in the centre of the cornea of the squinting eye. He then reads off the angle of the squint on the arc. If extreme accuracy is desired, the angle $\gamma$ should be measured and allowed for. It is done by covering the sound eye and making the patient fix the fixation spot of the perimeter with the squinting eye. The light is again carried along the arc until the corneal reflex is again in the centre of the cornea. The angle $\gamma$ is read off on the arc. The method is not suitable for children, and can only be employed with intelligent adults. One objection to it is the tendency to fix the ordinary fixation spot of the perimeter instead of a distant spot in the same line of vision. If this mistake is made errors arise, more particularly because the amount of dynamic convergence exerted in these cases is not always the same as with normal eyes.

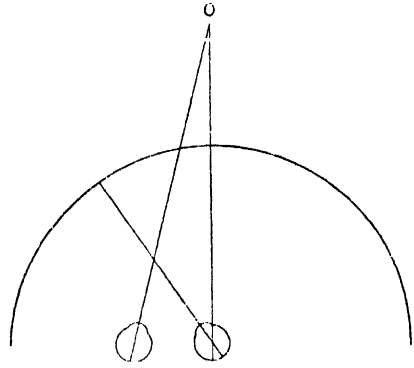



FIG. 317.—Measurement of the angle of squint by the perimeter.

The angle of deviation can also be conveniently measured on a tangent scale set against the wall, the corneal reflex of a light being again used as a guide.

*Treatment.* The routine treatment of a case of concomitant convergent strabismus in a child is as follows :—

(1) *Preliminary.* Record the distant vision of each eye if the child is not too young, the angle of the deviation, &c. Order ung. atropinæ, 1 per cent., three times a day for at least four days. At the end of this period estimate the error of refraction by retinoscopy and confirm the result subjectively if possible; reliance should be placed on the retinoscopy rather than on subjective tests. Again measure the angle of the squint, which is likely to be less under atropine than without a mydriatic. Order the full correction for constant use. A smaller correction for the effect of atropine should be made than in hypermetropia without squint. If the error is considerable I usually subtract only 0·5 D for atropine instead of 1 D; if the error is small I order the full atropine correction to start with. Great care must be taken to correct all astigmatism, especially in the squinting eye. The patient should be re-examined in a month's time.

If the child is less than two years old I do not order glasses except in rare cases. Some surgeons order them in all cases. I prefer to eliminate accommodation by keeping both eyes under the influence of atropine; the 1 per cent. ointment need be applied only once a day. The child should be examined at regular intervals until it is considered advisable to order glasses.

(2) *Occlusion of the Fixing Eye.* If, when the vision is tested, the squinting eye, as is usually the case, is amblyopic, an effort should be made to improve the vision in it by continual exercise. In order that this eye may be used the other must be prevented from seeing, or at any rate from seeing clearly. The only satisfactory method of ensuring this is by complete occlusion, which is effected by a patch of isinglass plaster, shaped so as to cover the better eye. A second smaller piece is applied to the adhesive side of the larger piece so that the smooth surface is towards the lids and an adhesive margin, half an inch wide, is left round the edge. This is stuck down to the temporal region, the forehead, the bridge and side of the nose, and across the cheek; over the malar bone a small air vent is left. The plaster is changed when it becomes dirty or loose. *Occlusion should be absolute*, and may have to be continued for six to twelve weeks or until the vision has

improved to 6/12 or 6/9, when the visual acuity is sufficient for orthoptic training. The child is examined at intervals of a month or two, and any improvement in vision in the squinting eye, as well as any change in the angle of deviation, carefully recorded. In some cases the deviation becomes transferred to the occluded or atropized eye: this is a good sign, as it indicates that the vision with the originally squinting eye is only slightly worse than that of the fixing eye.

In very young children or in recent squinters in whom the habit of suppression has not become fixed, a less drastic procedure may be sufficient. The best method is to order the instillation of atropine *into the fixing eye only*, once a day. This eye will then generally be used only for distant vision, the squinting eye being used for seeing near objects.

(3) *Orthoptic Training.* The further treatment depends upon the size of the angle of deviation, the condition of vision in the squinting eye, and a variety of other factors which differ in each case. An attempt is made to cultivate binocular vision and stereoscopic fusion by *orthoptic training.* This consists essentially in specially devised exercises. It has one overwhelming argument in its favour, viz., that when successful it cures the squint. The cure is complete. *i.e.*, the patient is placed in the same condition as a normal person; his eyes are straight and he has binocular vision. No other treatment can be said to cure the disorder. The eyes can be put straight, but this cures only the deviation; the other elements of the disorder remain unaffected.

There are three stages in orthoptic treatment: (1) the production of simultaneous vision with the two eyes, *i.e.*, the unmasking of diplopia; (2) the production of binocular vision, *i.e.*, the fusion of two halves of the same object presented simultaneously to the two eyes respectively; and (3) the production of stereoscopic vision, *i.e.*, the fusion of two images of the same object seen in perspective, resulting in the perception of relative distance of parts, solidity, and relief. The second stage will be facilitated if the two images are close together, and hence orthoptic treatment may demand operative treatment at an early age.

Unfortunately orthoptic treatment is extremely tedious and requires prolonged and very perservering efforts. Although it is of great value in inculcating correct visual habits such as the training of binocular vision and the abolition of false projection, it rarely if ever cures a squint of over 10° deviation or one of long standing. In many cases it is useless to attempt

it, and in all cases it is useless unless carried out systematically and thoroughly. For the details of the treatment monographs on the subject must be consulted. It has been considerably elaborated of recent years, and very encouraging results have been obtained, particularly when it is used as an adjunct to operative treatment. Few surgeons can spare the time to undertake it, but it should always be carried out under their supervision.

(4) *Surgical* treatment is indicated when the residual angle of squint is 10° or more when wearing correcting glasses, and in children between four and five years of age when orthoptic training has failed to bring the eyes parallel. As a general rule it should be undertaken as soon as the child is old enough to co-operate in post-operative orthoptic treatment.

Orthoptic training is an important preliminary to operation, and should be resumed as soon after as possible. When the angle of squint is 25° or more preliminary orthoptic training is generally waste of time, and operation should be undertaken early. The best results are in those operated on between four and six years of age. Postponement until the child is ten or more usually results in the permanence of amblyopia and failure to establish binocular vision. The operation is then purely cosmetic.

The safest operations for general use are an advancement or a recession of the appropriate muscle. Free tenotomy of the internal rectus tendon and its expansions into Tenon's capsule has often been followed in the past by divergence and retraction of the caruncle and plica semilunaris, owing to failure of reattachment to the globe. A tenotomy of the external rectus muscle, however, is permissible. The internal rectus should not be recessed more than 3·5 mm. lest weak convergence occur, leading to discomfort in reading and near work and to headaches. An internal rectus recession of 3·5 mm. will correct about 12° of squint. Considerable experience is required to assess the amount of recession and advancement needed in different cases.

If the deviation is 12° or less a recession of the internal rectus of the squinting eye should be performed. This will cure the deviation or reduce it to a negligible quantity.

If the deviation is more than 12°, advancement of the external rectus of the squinting eye, usually with recession of the internal rectus of the same eye, will be necessary. If the deviation is large advancement of the external rectus should always be accompanied by recession of the internal

rectus. This avoids an appearance of enophthalmos by allowing rotation of the globe approximately around the centre rotation of the eye. A general anæsthetic is to be avoided if possible, since the position of the eyes varies so much in different stages of anæsthesia that it gives no criterion of the final position after the anæsthetic has passed off. Very good results can, however, be obtained by an experienced operator if he keeps firmly in mind the amount of the deviation and ignores the position actually present under the anæsthetic. With pantocain only the operation is painless, except when tension is put upon the muscle. It is almost impossible to avoid slight tension during certain stages, but it need only be momentary.

In the case of young children a general anæsthetic should be used and any small residual deformity dealt with by orthoptic exercises.

The treatment of alternating concomitant convergent squint without appreciable error of refraction is purely cosmetic. These patients have no binocular vision, and it is useless to attempt to develop it unless the case is seen when the patient is very young, or immediately after the squint has been first noticed. Usually there is considerable deviation, so that an advancement operation is required. It should be postponed until a perfect result can be guaranteed, *i.e.*, until the operation can be performed with local anæsthesia.

In rare cases the patients develop diplopia after the eyes have been put straight. This may be due to false projection (*vide* p. 574), but also occurs with alternating squints. It is a very troublesome complication, since it usually persists for weeks or months, and is very distressing to the patient.

The treatment of concomitant divergent strabismus is similar to that of the convergent type. The refraction must be first carefully corrected, and it is advisable to order a full correction for constant use unless the myopia is very high. Tenotomy of the external rectus is seldom indicated in these cases because the benefit derived is too slight; it will not correct much more than 5° deviation. Hence advancement of the internal rectus is usually necessary. No operation is advisable in very high grades of myopia, since the size of the eye may render success mechanically impossible (*vide infra*).

In divergent strabismus slight over-correction is indicated, for these eyes show a great tendency to revert to their former position. In convergent strabismus the deviation should be fully corrected only in adults, on account of the tendency of

the deviation to diminish *pari passu* with the loss of accommodation (*vide* p. 576). Moreover, slight convergence is less unsightly than divergence.

**Divergent Strabismus other than Concomitant and Paralytic.** There are cases of divergent squint which are not paralytic, nor are they strictly concomitant. Some are very nearly allied to the true myopic divergent squint and may arise from it, *e.g.*, the divergence of the worse eye after binocular fixation has been completely abandoned. Similarly the mechanical divergence of extreme myopia is neither concomitant nor paralytic.

Another form of divergence, allied to the unilateral divergence in unequal myopia, is met with occasionally in any form of anisometropia in which the difference in refraction between the two eyes is great. In these cases, which are seldom susceptible of full optical correction (*vide* p. 535), one eye only is used for fixation. It is not surprising, therefore, that the unused eye assumes a position of rest, which is usually one of divergence. It is more surprising that relatively few such cases of anisometropia develop a manifest squint. When a squint is developed the patient not infrequently complains of diplopia, which is the more trying the less the divergence. These cases are difficult to treat. Prolonged use of the fullest possible correction, aided by stereoscopic exercises, should be tried first. If it fails, tenotomy of the external rectus of the diverging eye may effect a cure in slight deviations. In other cases an advancement is indicated; in such cases extreme delicacy of manipulation is required. Operation undertaken for cosmetic reasons may sometimes induce or increase diplopia by approximating the true and false images; it is usually permanent in these cases and exceedingly distressing.

Finally, a blind eye diverges sooner or later.

### Operations on the Extrinsic Muscles

**Simple Tenotomy.** It is to be remembered that this operation can only be safely performed upon the external rectus. The result which can be expected is only a correction of 5°. Instruments required: speculum, fixation forceps, flat strabismus hook (Moorfields' pattern), tenotomy scissors. Local anæsthesia suffices in all but very young patients; a few drops of adrenaline (1 in 1,000) may be instilled with advantage.

The conjunctival sac having been douched, and the speculum

inserted, the conjunctiva is seized over the muscle and a vertical incision, 8–9 mm. long, is made with scissors. The tissue underlying the conjunctiva at the lower border of the muscle is then seized with the forceps and incised, care being taken to hold the scissors so that they are tangential to the globe. If this is properly done Tenon's capsule is opened. The hook is now taken in the right hand, which still retains the scissors, and the point is introduced into the opening in Tenon's capsule. It is passed backwards, then upwards between the muscle and the sclerotic. In this manœuvre the point of the hook must be kept firmly against the globe. The point of the hook appears at the upper border of the muscle. It is freed from any conjunctival or subconjunctival tissue which may cover it. The hook is drawn forwards until it lies close under the insertion of the muscle. The hook is transferred to the left hand. The point of one blade of the scissors is introduced along the hook below the tendon, and the muscle is divided between the hook and the sclerotic. The hook should be introduced again and moved about in such a manner that any attachments which remain may be caught up and divided.

The conjunctiva is sutured with silk sutures and a pad and bandage are worn for two days, the eye being freely irrigated with boric acid lotion.

With the exception of a small puncture in the conjunctiva the whole operation can be performed subconjunctivally by an expert. It is less easy and slightly more dangerous.

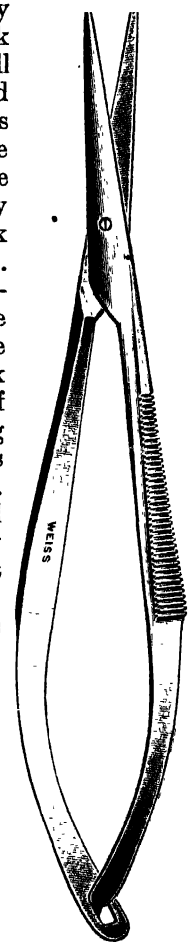



FIG. 318.—Spring scissors.

*Recession of the Internal Rectus.* Instruments required: Lang's speculum (Fig. 118), 2 pairs of conjunctival block forceps (Fig. 119), fixation forceps, 1 c.c. hypodermic syringe, 1½-inch hypodermic needle, spring scissors (Fig. 318), needle holder (Fig. 319), 3 sutures of 000 black silk on conjunctival needles, 6 bull-dog forceps for clamping sutures, 4 mosquito pressure forceps for gauze swabs, straight

probe, spirit lamp, 2 strabismus hooks (Fig. 271), pair of dividers, steel rule graduated in millimetres, mapping pen with terminal 3 mm. bent at right angles, gentian violet for marking, closed tubes of six 0 catgut sutures on eyeless needles, and No. 1 black sutures on eyeless needles.

A curved incision is made with scissors in the conjunctiva with its convexity towards the cornea, over the internal rectus. The flap is undermined by passing scissors under it towards the inner canthus, the blades being then opened ("spreading"). The subconjunctival tissue is pushed towards the nose with a gauze swab, and the muscle, covered by Tenon's capsule, exposed. Tenon's capsule is then seized with forceps just above and below the insertion of the muscle and button-holed with scissors. The capsule is slit for 7 mm. along the upper and lower edges of the muscle. Any bleeding points are touched with a probe heated in the flame of the spirit lamp. Tenon's capsule covering the muscle should be preserved. The point of a strabismus hook is passed into Tenon's capsule at the posterior limits of the incisions and retracted. Dividers measuring the amount desired to set the muscle back are placed along the upper and lower borders of the muscle, the distance measured off from the tendon insertion, and marked on the sclera with the mapping-pen. Two 000,000 catgut sutures on eyeless needles are passed through the upper and lower edges of the muscle 2 mm. behind its insertion in the so-called "whip stitch" fashion (Fig. 320). The tendon is divided, and the stitches are passed through the superficial layers of the sclera at right angles to the long axis of the muscle at the limit already marked. They are tied, and the conjunctival incision is then closed with silk sutures.

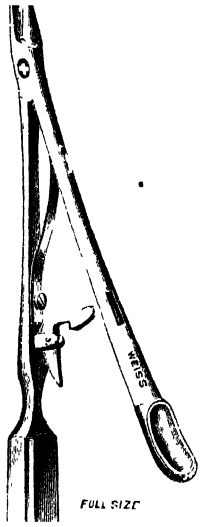


FIG. 319. — Silcock's needle holder.

**Advancement of the External Rectus.** An incision is made 2 mm. behind and concentric with the corneoscleral junction in front of the insertion of the muscle. The conjunctiva is then undermined by "spreading" (*vide supra*). The muscle is exposed in the same manner as in recession (*vide supra*). A strabismus hook is passed between the muscle and sclera, and the length of muscle and tendon for resection marked off (1 mm. advancement corrects about 2·5° deviation). No. 1 black silk whip-stitch sutures are passed through the upper

and lower edges of the muscle 2 mm. behind the gentian violet mark and ensnaring a breadth of 2·5 mm. of the muscle fibres.

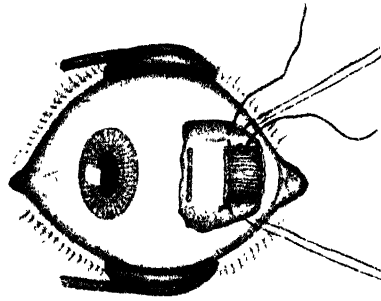


FIG. 320.—Recession of internal rectus

The muscle is divided at the mark, the distal part being held in fixation forceps so as to steady the globe whilst the scleral sutures are inserted. The eyeless needles carrying the whip-

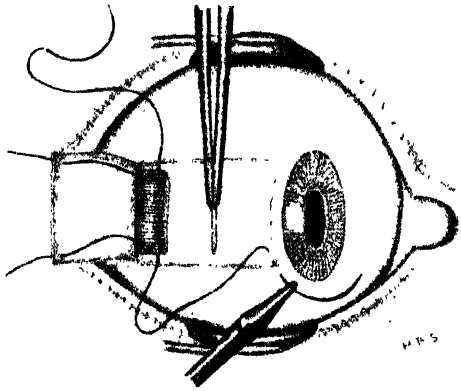


FIG. 321.—Advancement of external rectus.

stitch sutures are passed through half the thickness of the sclera transversely to the long axis of its fibres at the anterior marked spots (Fig. 321). It is essential to obtain a firm hold on the sclera, and in order to do this the needle is first passed

almost vertically for 0·5 mm., and then turned slightly towards the surface. It is driven through about 1 mm. by pressure *in the line of curvature of the needle.* The tendon is then divided at its insertion, the shortened muscle drawn forwards and the sutures tied. All blood is swabbed up, and the conjunctival incision closed with black silk sutures.

### Congenital Defects

One or more of the extrinsic muscles may be absent as a congenital defect, or may be abnormally inserted into the sclerotic. In some cases the condition has been proved to be due to the absence of the motor nervous mechanism. The position of the eyes and their movements may be very varied, but sometimes resemble those of an ordinary internal squint. In the latter case peculiarities of movement, *e.g.*, in and up instead of inwards, can usually be elicited. When each eye is made to fix successively, the movements of the eyes are often quite different from each other. There is never double vision and muscular contracture does not occur. There is often congenital ptosis (*q.v.*) not infrequently of the hereditary type, and sometimes nystagmus.

### Latent Strabismus or Heterophoria

It is found in some apparently normal persons that in the screen test (*vide* p. 553), when the screen is removed from before one eye, that eye moves slightly inwards or outwards to regain binocular fixation; if the screen is placed in front of the other eye and then removed, this eye also moves slightly inwards or outwards respectively to regain fixation. When both eyes are fixing there is no deviation. Such a squint is called a latent squint or, in opposition to the normal condition of orthophoria, heterophoria. If the latent squint is one of convergence the condition is called esophoria, of divergence exophoria. Sometimes one eye is higher than the other; this condition is usually called hyperphoria: as a matter of fact it is impossible in these cases to be sure whether there is absolute hyperphoria of one eye or hypophoria of the other, the condition being relative only.

It must be concluded that when the eyes are screened they take up a position of rest, the extrinsic muscles exercising merely the tone normal to them at the time of examination. In cases of latent squint the position of rest is not orthophoria, with the visual axes parallel, but heterophoria, with some deviation of the axes. During normal vision the requirements

of binocular vision demand a suitable readjustment of the visual axes, which can be brought about only by tonic contraction of certain muscles—in esophoria, of both external recti, in exophoria, of both internal recti. This involves a perpetual strain, which often manifests itself as asthenopia. As might be expected the deviation is liable to become manifest in conditions of bodily fatigue and to vary in amount from time to time. Some periodic squints are due to this cause, and the periodicity may be rhythmic. Thus a child may squint in the evening when he is tired; after a good night's rest the squint has disappeared, and may not return until the second or third day, the sequence being accurately repeated. Often latent squints give no trouble until school time arrives or adult life is reached. Here the demands of near vision increase the strain. No symptoms arise perhaps until after reading or writing for an hour or two. Then "the letters seem to run together." This is due to relaxation of the over-strained muscles; the eyes momentarily assume the position of rest, and diplopia, which is not realised as actual double vision, causes blurring of the print. With an effort the blurring is overcome, but eventually this becomes impossible, headache supervenes, and the work has to be abandoned.

Analysis of the cases shows that slight eso- and exo-phoria are quite common and give rise to little or no trouble, which is not difficult to understand when it is remembered that over-action of both internal recti is physiological in ordinary convergence on near objects. These muscles are therefore accustomed to act together and little strain is felt. The same is true in less degree of the external recti. Only when the deviation is great —5° to 10° or more—is asthenopia frequently present. Very slight degrees of hyperphoria, however, almost invariably cause extreme discomfort, for in these cases over-action of muscles which are not accustomed to work together is necessary in order to keep the visual axes in the same plane. For instance, in the primary position of the eyes there must be over-action of one superior rectus and inferior oblique, combined with over-action of the other inferior rectus and superior oblique, and the

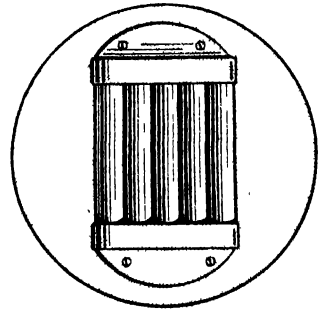


FIG. 322.—Maddox rod.

readjustment in other positions of the eyes must be very complex.

It may be impossible to discover slight degrees of heterophoria by the screen test. More delicate tests have therefore been devised. All depend upon disassociating the two eyes. The simplest method is that of so altering the appearance of the retinal image in one eye that it affords no stimulus to fusion with the image of the other eye.

*The Maddox Rod Test.* The patient is placed six metres from a candle or bright spot of light in a dark room. A Maddox rod (Fig. 322), which consists of four or five cylinders of red glass side by side in a brass disc, is placed in the trial frame before one eye. The spot of light seen through the red cylinders appears as a long red line. If the cylinders are placed with their axes horizontal the red line will be vertical. If there is orthophoria the bright spot will appear to be in the centre of the vertical red line; if there is eso- or exo-phoria the red line will be to one side of the spot. The angle of the deviation is measured by the strength of the prism which it is necessary to place in front of the Maddox rod in order to bring the red line and the spot together. The nature of the deviation is indicated by the position of the base of the prism, whether out or in.

The Maddox rod is then turned round so that the cylinders are vertical; the red line will now be horizontal. If there is no hyperphoria the line will pass through the bright spot. If there is hyperphoria the red line will be below or above the spot according as the relative hyperphoria is in the eye with the rod in front of it or in the other. The amount of deviation is measured either on a tangent scale or by the strength of the prism required to correct it.

*The Red-Green Test.* A vertical slit covered with red glass above and green glass below is illuminated from behind, and viewed by the patient through reversible spectacles containing a red glass in one eye and green in the other. The glasses are chosen such that the red glass absorbs all rays transmitted by the green, and *vice versâ*. With orthophoria the two lights are seen in their proper position; with heterophoria they are displaced, but may become replaced by muscular effort. Disappearance of one light indicates complete suppression of the image of the other eye.

*The Diaphragm Test.* In its original form in Rémy's diploscope letters are viewed on a stereoscope frame through two slits in a diaphragm interposed between the eyes and the

letters. In Bishop Harman's diaphragm test (Fig. 323) letters or numbers are viewed in a similar manner through a single central slit, the width of which is adjustable. The width of the slit is shown on an arbitrary scale, thus enabling a numerical record of the examination to be made (see p. 705).

The Red-Green Test and the Diaphragm Test have been adopted by the Royal Air Force for their examination of candidates (*vide* p. 704), for it has been found that latent squint is a potent cause of bad landings.

The deviation in latent squint is often different in near vision from that in distant, so that both must be tested. The

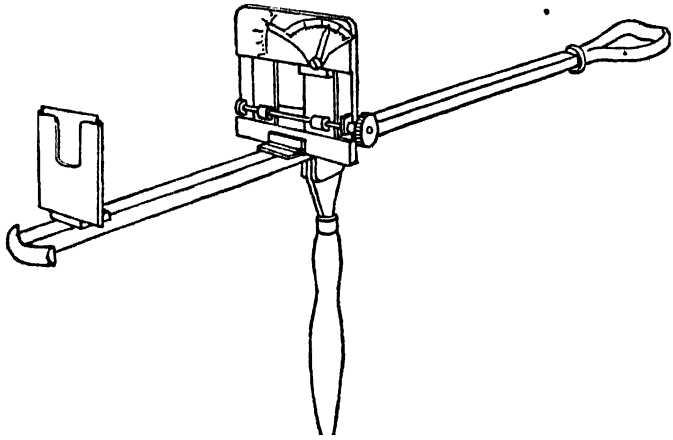


FIG. 323.—Bishop Harman Diaphragm Instrument.

deviation in near vision is tested by means of a special card (Plate XXI). A strong prism—about 12°—is placed base down or up before one eye. In orthophoria the arrows are exactly in the same vertical line. In eso- or exo-phoria the lower arrow points to a number in the upper scale. This number gives the angle of deviation, which may be confirmed by counteracting the deviation with a prism, base in or out, before one eye. The Maddox wing-test or the Bishop Harman diaphragm test (*vide* p. 705) is a convenient method of applying this test.

Besides the actual measurement of the deviation in latent strabismus the strength of the muscles involved should also be tested by forcing them to a maximum effort against prisms. With the patient seated six metres from a candle the highest

prism, base down before the right eye, still permitting of single vision, gives the range of superduction of that eye. Subduction and abduction can be measured in the same manner. Adduction gives less concordant results. The normal limits of super- and sub-duction are 1·5° to 2·5°; of abduction 4° to 5°.

Allied to these defects, though not strictly speaking a latent squint, is insufficiency or weakness of convergence. It will be revealed by the ordinary tests of motor balance. If there is more exophoria or less esophoria in near vision than in distant there is insufficiency of convergence. The majority of cases have orthophoria or esophoria for distance, but exophoria for near work. Most of them have hypermetropia, but it is not uncommon in myopes. Simple tests for convergence are described in the examination of candidates for the Royal Air Force (*vide* p. 704). The defect causes asthenopia in near work.

*Treatment.* The lower degrees of esophoria, and to a less extent of exophoria, cause no symptoms and need no special treatment. Slight exophoria often causes symptoms in young adults much engaged in near work. It is relieved by suitable prisms, bases in; if the general health improves, or the amount of near work is diminished, the prisms can be dispensed with later. Hyperphoria is most likely to cause asthenopic symptoms. It is corrected by ordering suitable prisms to be combined with the glasses which correct any refractive error. If the spherical error is sufficiently great the prismatic effect may be obtained by decentring the lenses. The total prismatic error should be divided equally between the two eyes in ordering the correction. Thus, if there is hyperphoria of 3° as measured by a 3° prism, base down before the left eye, a prism of 1½° is ordered before each eye, base up for the right, base down for the left. When this treatment does not succeed, and the deviation is considerable, tenotomy of the superior rectus may be necessary, but such cases are rare, and operative interference should not be lightly undertaken, for it is apt to be disappointing.

The rational treatment of large degrees of eso- or exo-phoria consists in exercising the weak muscles against prisms. This is usually only temporarily beneficial, but the muscles can be kept in good order by repeating the exercises at intervals. The asthenopia can be relieved by ordering prisms to correct the defect, *i.e.*, prisms with their bases directed in the opposite sense to those used for exercise. This should be avoided except in such cases as those already mentioned, since it

generally tends to increase the defect, so that stronger prisms have to be ordered from time to time. In severe cases a course of orthoptic exercises should be given and operative interference may be indicated.

Insufficiency of convergence may be treated by prism exercises. The following simple exercise is often sufficient without having recourse to prisms. Any error of refraction is corrected with glasses which are ordered to be used constantly. While reading the patient gradually brings the book nearer and nearer, until the print becomes blurred. He then slowly moves the book back to ordinary reading distance. The process is repeated. At about every tenth line the patient looks into the distance, so as to relax his accommodation and convergence. Two or three pages should be read in this manner three or four times a day for several weeks. The course is repeated as often as necessary. More effective are orthoptic exercises with stereoscopic apparatus.

If convergence training fails prisms, base in, must be ordered with the reading glasses. Care must be taken not to overcorrect presbyopia (*vide* p. 539).

# SECTION VI

## SYMPTOMATIC DISEASES OF THE EYE

### CHAPTER XXIX

### Ocular Manifestations of Diseases of the Nervous System

MANY diseases which primarily attack other parts of the body give rise to ocular symptoms, and not infrequently first come under the observation of the ophthalmic surgeon. At the risk of some repetition I propose briefly to review the most important ocular manifestations of such diseases.

The ocular signs of nervous disease often appear superficially to be complicated and confusing. In most cases they are readily explained by the anatomy of the part of the nervous system involved.

**Tabes Dorsalis.** *Primary Optic Atrophy* (*vide* p. 401) occurs in about 10—20 per cent. of cases of locomotor ataxia. It is about twice as common in men as in women, most frequent between thirty and fifty years of age, and may precede the appearance of typical tabetic symptoms by some years. It is commonest in the pre-ataxic stage, but it is not true that optic atrophy deters the development of ataxy or exercises any beneficial influence. The onset is gradual, leading to total blindness in two to three years or more. Pallor of the disc may precede the failure of vision by a considerable period, never the reverse. The affection of one eye usually precedes that of the other by a few months, rarely longer. The pathology is not definitely established, but it would appear that the disease is probably a primary interstitial neuritis arising in an extremely chronic exudative process from the pia and causing a secondary degeneration of the nerve fibres and their parent ganglion cells. The process usually becomes apparent first in the intra-cranial portion of the nerve distal to the chiasma.

The *fields* show progressive contraction, *pari passu* with the failure in central vision. It is rare for the failure of sight to commence with a central scotoma, thus differing from the

onset in disseminated sclerosis, though Fuchs has shown that it occurs (*vide* p. 404). Two types of field are met with: (1) General concentric shrinkage, the colour fields for red and green being very early lost, and central vision much impaired; (2) Irregular sectorial defects, which are sharply defined but

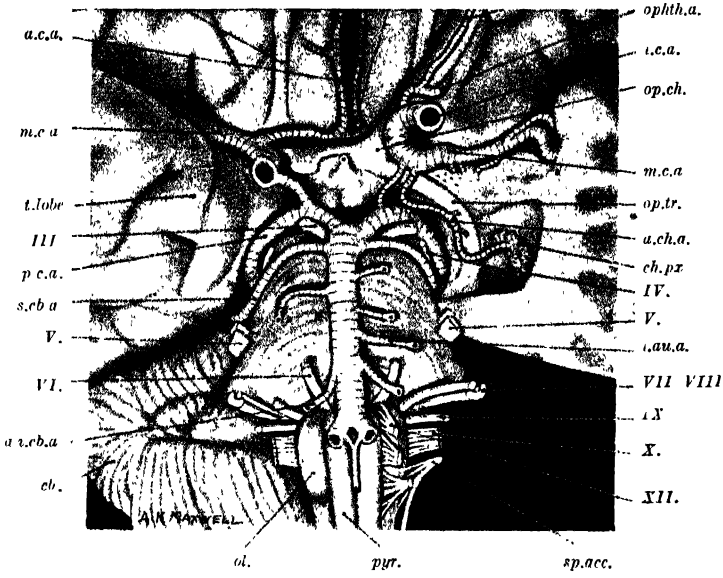



FIG. 324.—Relations of the cranial nerves to the arteries at the base of the brain. *a.c.r.*, arteria centralis retinæ; *ophth.a.*, ophthalmic artery; *i.c.a.*, internal carotid artery; *op.ch.*, optic chiasma; *a.c.a.*, anterior cerebral artery; *m.c.a.*, middle cerebral artery; *op.tr.*, optic tract; *a.ch.a.*, anterior choroid artery; *ch.px.*, choroid plexus; *i.au.a.*, internal auditory artery; *sp.acc.*, spinal accessory nerve; *pyr.*, pyramid; *ol.*, olive; *cb.*, cerebellum; *a.i.cb.a.*, anterior inferior cerebellar artery; *s.cb.a.*, superior cerebellar artery; *t.lobe*, temporal lobe. (Eugene Wolff. "Anatomy of the Eye and Orbit." Lewis, London.)

gradually spread, though central vision may be quite good. Defective dark adaptation and coloured vision have been described as early symptoms of tabes.

The characteristic *pupillary signs* include the so-called spinal miosis, the Argyll Robertson pupil reaction, inequality of the pupils, and distortion of the pupillary aperture. These signs are found in other diseases, and are to be regarded as signs of syphilis of the central nervous system rather than

as pathognomonic of tabes. Their combination is of great diagnostic significance. Argyll Robertson pupils are found in 70 per cent. of tabetics and are almost invariably bilateral. Unequal pupils are found in 30 per cent. of tabetics, but are still more frequently met with in general paralysis of the insane. Ophthalmoplegia interna, *i.e.*, paralysis of the sphincter iridis and of the ciliary muscle, occurs in about 5 per cent. of tabetics and is generally unilateral. It is due to a lesion in the nucleus of the IIIrd nerve. Cycloplegia without mydriasis, or *vice versâ*, is rare.

*The Myotonic Pupillary Reaction* resembles the Argyll Robertson pupil, but occurs in the absence of all signs of syphilis. It is characterised by inaction of the pupil to the light stimulus and slow reaction and recovery to convergence (Foster Moore). It is usually unilateral. Tendon reflexes may be normal or impaired. It differs from the true Argyll Robertson pupil in the sluggishness of the convergence reaction, and the fact that the tonic pupil is always larger than its fellow, whereas the unilateral Argyll Robertson pupil is always smaller (Adie). It is commoner in women than in men.

*Paralyses of the Extrinsic Ocular Muscles* are common in tabes, occurring in about 20 per cent. of the cases. The order of frequency of the nerves affected is IIIrd (20 per cent.), VIth (13 per cent.), IVth (3 per cent.), external or total ophthalmoplegia (2 per cent.). It is characteristic of tabetic paralyses that they are partial, *i.e.*, not involving the whole nerve, incomplete, *i.e.*, pareses rather than paralyses, variable and transitory. The affection of the IIIrd nerve is so common that it is always suggestive of a tabetic or syphilitic lesion. Total IIIrd nerve paralysis is rare in tabes, and isolated ptosis is very common (4 per cent. of cases). The pareses of the ocular muscles nearly always occur in the pre-ataxic stage: when they occur at a later stage they are more likely to be permanent. They generally clear up rapidly, but show a marked tendency to recur. They may be due to nuclear lesions, or to involvement of the proprioceptive fibres (Sherrington). Considering that tabes is essentially a disease of afferent tracts it is curious that the Vth nerve almost always escapes. Nystagmus is rare, but the paresed muscles often give rise to jerky movements of the eyes which may be mistaken for nystagmoid jerks. Paralyses of associated movements, *e.g.*, conjugate deviations, are very rare.

In **Combined Sclerosis**, in which both the posterior and the

lateral columns of the cord are affected, all the ocular manifestations characteristic of tabes may occur; these are probably atypical cases of tabes. In subacute combined sclerosis, such as is specially associated with anæmia and cachexia, ocular symptoms are rare.

In **Hereditary Ataxy** (*Syn.—Friedreich's Disease*) optic atrophy and paralyses of the ocular muscles are very rare. Nystagmoid jerkings of the eyes, very similar to those occurring in disseminated sclerosis, are very common, but the visual symptoms characteristic of the latter disease are absent. The movements are probably due to the same lack of co-ordination which causes the other ataxic signs of the disease; they occur on voluntary movement, and are not usually present in passive fixation.

**Congenital Spastic Paralysis** (*Syn.—Little's Disease*) is probably due to subdural hæmorrhage resulting from difficult labour. Of these cases 30—40 per cent. have concomitant convergent squint.

**Neuro-myelitis Optica** (*Syn.—Devic's Disease*). A considerable number of cases of optic neuritis associated with myelitis have been described. The visual defect usually precedes the signs of myelitis. Its onset is sudden, but one eye may be affected a day or so before the other. Complete amaurosis generally supervenes rapidly. In some cases there is a central scotoma in the early stages, and there may be pain on moving the eyes, pointing to a retrobulbar neuritis. There is usually only slight neuritis, but considerable swelling of the disc has been seen. In cases which recover the blindness passes off and good vision is restored. The site of the myelitis may be lumbar or dorsal, and the affection is due to a demyelinating virus. There are no signs of general meningitis, and other cranial nerves escape.

During the amaurotic stage the pupils are dilated and immobile. In cases of cervical and upper dorsal myelitis without optic neuritis the pupils are often unequal, owing to implication of the dilatator tracts. In these cases the pupils still react to light.

**Disseminated Sclerosis** (*Syn.—Multiple or Insular Sclerosis*). Lesions in disseminated sclerosis often occur in the visual paths (50 per cent. of cases) (Fig. 325). Unlike the lesions of tabes, the medullary sheaths of the nerve fibres are specially attacked, the axis cylinders remaining relatively little affected. Hence, during the acute stage, defects in conductivity are specially prominent; considerable variations succeed each

other, and high degrees of functional restoration are possible. The optic nerves are most frequently attacked, with all the clinical signs of a typical retrobulbar neuritis, but patches of degeneration in the chiasma, optic tracts, or optic radiations may cause characteristic hemianopic or quadrantic changes in the fields. In ordinary cases there is central scotoma with a full field. The scotomata are generally only relative: they

FIG. 325.—Disseminated Sclerosis. From a specimen by Gordon Holmes, photographed by Coats. Chiasma, optic nerves and tracts. Stained by Weigert-Pal method, the light areas being patches of degeneration.

are therefore easily overlooked, and can be demonstrated only by the use of small coloured objects. Concentric contraction of the field and irregular peripheral defects, sometimes only for colours, also occur, and these show variations from time to time. Hemianopic fields are rarer than might be expected.

The ophthalmoscopic changes show very little direct relationship to the visual defects. There may be complete blindness with normal fundi, and signs of optic atrophy may

coincide with good vision. Owing to the relative escape of the axis cylinders much less peripheral degeneration occurs than might be anticipated. Owing to the recovery of conductivity in the fibres, vision generally improves materially, but repeated relapses are not uncommon. The visual symptoms may precede other signs by several years. Permanent complete blindness scarcely ever occurs. Uhthoff found marked optic atrophy in 3 per cent., incomplete atrophy in 19 per cent., temporal pallor of the disc in 18 per cent. and optic neuritis in 5 per cent. of cases. The optic nerves are affected, therefore, much more commonly than in tabes, more often indeed than in any other disease of the nervous system except cerebral tumour.

The visual symptoms of disseminated sclerosis may be mistaken for those of toxic amblyopia, retrobulbar neuritis, tabes, or hysteria. The diagnosis from the two former is the most difficult. In ordinary retrobulbar neuritis the central scotoma is usually absolute; in disseminated sclerosis relative. In toxic amblyopia the scotoma is practically always bilateral; in disseminated sclerosis it is unilateral in about half the cases. In retrobulbar neuritis it is nearly always unilateral, and to these cases the early stages of disease of the pituitary gland must be added. Some cases can only be definitely diagnosed by the history and by the development of other pathognomonic signs. With regard to tabes central scotoma is rare, and on the other hand symmetrical concentric contraction of the field is rare in disseminated sclerosis. Moreover, the failure of vision is steadily progressive in tabes, and is bilateral; in disseminated sclerosis it is unilateral and very variable. The diagnosis from hysteria may be difficult, but the regular concentric contraction of the field so often found in this condition scarcely ever occurs in disseminated sclerosis and lack of sustained contraction of the pupil to light (*vide* p. 395) is pathognomonic of organic disease.

Nystagmus occurs in multiple sclerosis (12 per cent. of cases), but nystagmoid jerks are much commoner (50 per cent. of cases). True nystagmus is a very important diagnostic sign, as it is rare in other acquired diseases of the central nervous system: nystagmoid jerks are of much less diagnostic significance. Both are probably due to central changes, and the latter show some analogy to the intention tremor so characteristic of disseminated sclerosis.

Miosis is fairly common in this disease, and to a less degree

inequality of pupils. Other abnormal pupil reactions are rare.

Paralyses of extrinsic ocular muscles are much less common than in tabes, and although resembling these in their partial and transitory nature differ from them in that paralyses of associated movements are not uncommon. Thus paresis of convergence, with retained action of the recti in lateral movements, frequently occurs. Paralysis of lateral conjugate movements is commoner than that of upward and downward movements. These are obviously due to nuclear or supranuclear lesions. Of individual nerves the VIth is more often affected than the IIIrd, and total IIIrd nerve paralysis is never seen (cf. Tabes). Partial ophthalmoplegia externa, with intact intrinsic muscles, also occurs, whereas ophthalmoplegia interna is unknown.

**Syringomyelia** is due to dilatation of the central canal of the cord and excavation of the central grey matter. Since the dorsal and lower cervical regions are often affected inequality of the pupils is the most characteristic ocular sign. It is due to implication of the dilatator tract; the pupil on the affected side is small and reacts to light, but does not dilate after instillation of cocaine (*vide* p. 63). Other signs of paralysis of the cervical sympathetic may be present, such as slight ptosis, retraction of the globe, &c. Paralysis of the Vth nerve is also not uncommon, and the VIth nerve may be affected, but very rarely the IVth or IIIrd. Syringomyelia, however, is sometimes complicated with tabes, and in these cases all the ocular manifestations of tabes may occur. The patients are often hysterical, a fact which accounts for the frequency of concentric contraction of the fields of vision.

**Myasthenia Gravis.** This disease shows some resemblance to chronic progressive bulbar paralysis, but differs from it, *inter alia*, in the fact that the ocular muscles are almost invariably affected. Most of the patients are young, and have difficulty in articulation, swallowing, and mastication. There is nearly always ptosis and paresis of the orbicularis palpebrarum. The muscles of the extremities and trunk become affected, or the disease may start in them. Dyspnœa is common, especially on exertion. A striking feature is the absence of muscular atrophy. The muscles do not give the reaction of degeneration, but show the "myasthenic reaction," *i.e.*, they respond worse and worse to repeated faradic stimuli. The most characteristic feature is the rapid fatigue of the muscles. The symptoms are least marked in the morning,

*e.g.*, the ptosis is much worse in the evening. Reading is only possible for a few minutes owing to failure of convergence and lateral movements of the eyes. Only a few mouthfuls of food can be masticated owing to fatigue of the muscles. The same applies to other voluntary muscles, which are similarly rapidly tired out by electrical stimulation. The muscles recover rapidly in the early stages of the disease after a short rest. The symptoms fluctuate from day to day, and may remain in abeyance for considerable periods. Sensory and cerebral symptoms are absent, and the reflexes are normal. Many cases die of failure of respiration, though the course of the disease is usually long. No pathological changes are found in the nervous system, but groups of lymphocytes ("lymphorrhages") have been found in the muscles, and the thymus is sometimes enlarged.

The ptosis is nearly always bilateral and is increased by prolonged fixation or attempts to look upwards. Effective compensation by over-action of the frontales is impossible. Ophthalmoplegia externa, partial or complete, occurs in 50 per cent. of the cases. The intrinsic muscles are not affected. Nystagmoid jerks are not uncommon.

Remarkable temporary improvement in the action of the muscles is obtained by injections of prostigmin.

**Myotonia Atrophica** is a familial, hereditary disease, characterised by weakness of muscles (facial, vasti, &c.), and slow relaxation after contraction. The patients frequently develop cataract at an early age—20 to 40—and the cataracts may be the first manifestation of the disease.

**Ophthalmoplegia.** Ophthalmoplegia is a somewhat indefinite term applied to widespread paralysis of the muscles of the eye which is thought to be due to nuclear lesions and forms the most prominent feature of the cases. Nuclear paralyses often cause defects of convergence, conjugate deviation of the eyes, and so on; when these are isolated signs the term ophthalmoplegia should not be applied to them. In typical ophthalmoplegia both eyes are affected, though all the muscles need not necessarily be simultaneously paralysed. In ophthalmoplegia totalis all the muscles, extrinsic and intrinsic, are affected: in ophthalmoplegia externa, many or all of the extrinsic muscles; in ophthalmoplegia interna, the intrinsic muscles. Ophthalmoplegia may be acute or subacute and chronic.

Acute or subacute ophthalmoplegia is usually due to poisons

or infection, and is relatively rare. The chief poisons are alcohol, lead, and ptomaines; the chief infections, diphtheria and influenza. In thiamine deficiency (*vide* p. 698), frequently associated with alcoholism, the onset is sudden and accompanied by cerebral symptoms—headache, delirium, coma, &c. Bilateral ophthalmoplegia externa comes on suddenly or rapidly, with or without ptosis, and is often followed by facial and bulbar paralysis, with difficulty in speech and swallowing. The intrinsic muscles usually escape. Pathologically the condition is an acute hæmorrhagic superior polioencephalitis (Wernicke). In lead poisoning the onset is less acute and the intrinsic muscles are more often involved. In ptomaine poisoning, due to bad food, mussels, &c., the essential feature is bilateral ophthalmoplegia interna, with or without ptosis, but total ophthalmoplegia also occurs. In diphtheria isolated ocular palsies are common, but ophthalmoplegia externa is rare. The pupil often escapes, the ciliary muscle never. In influenza the ophthalmoplegia resembles that of diphtheria—extrinsic muscles and ciliary muscle, the pupil escaping, but the pupil has been known to be affected without the ciliary muscle. The prognosis in hæmorrhagic lesions is bad: other cases usually recover.

Chronic ophthalmoplegia is usually progressive. It commences with ptosis or diplopia. In the course of months or years the paralysis spreads to all the ocular muscles of both sides, except that the intrinsic muscles often escape, and not infrequently the levatores palpebrarum also. These cases of isolated chronic ophthalmoplegia are rare, but the condition is often a precursor or symptom of tabes or general paralysis of the insane, rarely of disseminated sclerosis, &c. It is a very early sign of tabes, and may become associated later with bulbar symptoms. The Argyll Robertson pupil or ophthalmoplegia interna is often present.

Ophthalmoplegia also occurs as a congenital disease or may be acquired early in life as an hereditary familial disease. In these cases there is usually only partial ophthalmoplegia externa, and the condition is not complicated by other nervous disease such as tabes or bulbar paralysis, thus differing from the adult acquired form.

**Diseases of the Pons.** The ocular symptoms are of great localising value in diseases of the pons. Of these, tumours are by far the most common, hæmorrhages, thromboses, softening, and abscess being relatively rare.

*Tumours.* By far the commonest tumours of pons are the

tubercle and glioma, the former being about twice as common as the latter. Both occur most frequently in childhood. Papilloedema or papillitis occurs in about half the cases, and is accompanied by the usual visual symptoms. The most characteristic signs of pontine tumours are due to implication of the motor nuclei and pyramidal tracts (*vide* Figs. 290—296). The VIth nucleus is usually implicated and causes loss of conjugate movement of the eyes to the same side (*vide* p. 546) with retention of convergence: at the onset the external rectus only may be paralysed. Owing to the immediate vicinity of the pyramidal tract it is also generally involved, and as the fibres are affected before they decussate in the medulla oblongata there is contra-lateral hemiplegia. The intimate relationship of the VIIth nucleus and its afferent fibres to the VIth nucleus has already been mentioned. Hence, facial paralysis combined with loss of conjugate deviation of the eyes to the same side suggests a pontine lesion. Similarly facial paralysis with contra-lateral hemiplegia (Millard-Gubler's syndrome) has the same significance. If the lesion is situated high up in the pons the pyramidal tract is caught before the fibres to the facial nucleus have crossed. Hence in these cases there is facial paralysis combined with hemiplegia on the same side. The facial paralysis is then usually of the cerebral type, in which the orbicularis palpebrarum, which is said to be innervated from the IIIrd nucleus, escapes. Not infrequently the Vth nerve is partially paralysed, causing, for example, paralysis of the Vth, nuclear VIth, VIIth, and sometimes VIIIth nerves, with crossed hemiplegia. It is astonishing how large pontine tumours can become without causing death, the nervous structures being pushed aside, especially in the relatively slow development of tuberculous masses. Extension of the disease may lead to IIIrd nerve paralysis, practically never IVth, the fibres of which are protected by the dorsal position of their decussation. If ptosis is the only sign of involvement of the IIIrd nerve its localising value is slight; it may be a mere pressure symptom or a cerebral ptosis. Occasionally the opposite pyramidal tract is involved, with bilateral hemiplegia. Owing to the combination of facial paralysis the cases in which the trigeminal is involved are more likely to cause neuroparalytic keratitis (*q.v.*) than are other lesions of the Vth nuclei or intra-medullary fibres. Miosis is not uncommon in tumours of the pons, but the pupillary signs are of little diagnostic value; nystagmus is a sign of involvement of the cerebellum.

In *hæmorrhages* and *thromboses* in the pons the same motor signs are manifest, and are usually of rapid or sudden onset. There are no ophthalmoscopic changes. The pupils are usually very small in the early stages of pontine hæmorrhage, a point of considerable diagnostic significance in an unconscious patient.

**Tumours of the Auditory Nerve** (*Syn.—Extra-cerebellar Tumours*). The peculiar slow-growing neuro-fibromatous or endotheliomatous tumours of the recessus acustico-cerebellaris, usually attached to the VIIIth nerve, give rise to a fairly characteristic syndrome with ocular signs. Early deafness on one side is associated with cerebellar symptoms, among which nystagmus is common. The VIth nerve is usually involved, generally with paralysis of the external rectus only, rarely with paralysis of conjugate deviation. As might be expected, there is very often facial paralysis of the peripheral type, *i.e.*, total, including the orbicularis palpebrarum. The Vth nerve is implicated in about a quarter of the cases, but neuroparalytic keratitis is uncommon. In nearly all the cases there is pronounced papillœdema.

**Diseases of the Cerebral Peduncle** (Figs. 56, 324).—The most characteristic sign of disease of the cerebral peduncle is a combination of paralysis of the IIIrd nerve with contra-lateral hemiplegia, the latter including the face and tongue (Weber's syndrome). The facial paralysis is naturally of the cerebral type, in which the orbicularis palpebrarum escapes, since it is due to a pyramidal tract lesion. If the red nucleus (Fig. 291) is involved tremor and jerky movements occur in the contra-lateral side of the body: this condition, combined with ipsi-lateral IIIrd nerve paralysis, forms Benedikt's syndrome. Motor and sensory hemiplegia, contra-lateral to the lesion, without IIIrd nerve paralysis, is less common than Weber's syndrome, and IIIrd nerve paralysis alone is rare. In the usual syndrome the whole IIIrd nerve is involved, the intrinsic muscles rarely escaping: when it occurs it is due to an intra-peduncular fascicular lesion. Both oculomotor nerves are sometimes affected. As might be expected (Figs. 56, 291), implication of the external geniculate body or optic tract may occur, with development of homonymous hemianopia. Since the commonest lesion in this region is solitary tubercle, papillœdema occurs in about 10 per cent. of the cases. The most frequent other causes, omitting basal gummatous meningitis, which may affect the peduncle secondarily, are softening and hæmorrhage.

**Diseases of the Corpora Quadrigemina and Pineal Gland.** Though there can be no doubt that visual functions are located in the optic lobes of lower animals these functions are submerged in their later representatives, the anterior colliculi. There is no good evidence that lesions of the corpora quadrigemina cause any direct impairment of vision in man. The anatomical relations of the posterior colliculi point to association with hearing, and lesions of the corpora quadrigemina are frequently accompanied by impairment of hearing, which may, however, be due to pressure on the auditory paths. The commonest lesion of these bodies is solitary tubercle, which acts like an intracranial tumour, and glioma. Tumours of the pineal gland are generally gliomata; they press upon the colliculi and cause similar though less pronounced symptoms.

As might be expected, tumours in this region very frequently cause papillœdema from pressure on the aqueduct of Sylvius, and therewith deterioration of vision. The sign of greatest localising value is loss of upward and downward movement of both eyes. Sometimes only upward movement is lost, never downward alone. The other movements of the eyes are relatively good. It is noteworthy, in opposition to experimental data, that impairment of conjugate lateral movements of the eyes is almost unknown in these lesions. In more than half the cases there is paresis of both IIIrd nerves. Less often only one oculomotor nerve is affected. Extension of the pressure effects may lead to bilateral IVth nerve paralysis or ophthalmoplegia externa, but the VIth nerve, as might be expected, is seldom directly affected. Pupillary changes are common, owing to implication of the IIIrd nerves or papillœdema. Experimental and clinical evidence alike tend to show that, in spite of the intimate relationship of the afferent pupillary paths with the superior colliculi, lesions of these bodies cause no direct permanent changes in the pupillary reactions. The facial nerve is paralysed in about a quarter of the cases: the paralysis is of the cerebral type. It is occasionally accompanied by ipsilateral hemiplegia. These are distant signs, due to pressure. Nystagmus occurs more frequently than with other cerebral tumours, but is usually associated with defects of co-ordination and other signs of implication of the cerebellum. These cases are always difficult to diagnose from cerebellar lesions, and the order of onset of the symptoms is important. If the ocular movements are affected first, and especially if upward

and downward movements are lost, the lesion is probably quadrigeminal; if the cerebellar ataxy precedes the impairment of ocular movements the lesion is probably in the cerebellum (Bruns).

**General Paralysis of the Insane** (*Syns.* — *Progressive Paralysis, Paralytic Dementia*). Like tabes, this is a parasyphilitic disease. It is often accompanied by tabetic signs and symptoms which are due to lesions of the posterior tracts of the cord, identical with those in tabes (tabo-paralysis). The ocular symptoms are most common and unequivocal in these cases, and are to be attributed to the same causes.

The *pupillary* changes are most characteristic. In the early stages inequality of the pupils is most common. It should be quite definite to be of diagnostic value, for slight inequality is not very infrequent in normal people. It is often accompanied by slight deformation in the shape of the pupil and irregularity of the pupillary margin. The same remark applies to these changes. The pathological nature of the pupillary changes is put beyond doubt when there is the typical Argyll Robertson reaction. It occurs in nearly half the cases, and is therefore an important sign, but less constant than in tabes. In about 5 per cent. of the cases the reactions both to light and convergence are lost, a condition which is rare in tabes and especially frequent in the juvenile form of general paralysis. The sensory reaction, *i.e.*, dilatation of the pupil on painful stimulation of the skin, is very often lost with the light reaction. The Argyll Robertson pupil is rare in cases in which the knee jerks are retained. Spinal miosis is commoner in tabes, unequal pupils in general paralysis. Ophthalmoplegia interna is rarer in general paralysis.

*Primary optic atrophy* occurs in about 8 per cent. of cases (Uhthoff). It shows exactly the same type and course as in tabes, but is more frequent in the latter disease. Like the pupillary signs, it may precede the onset of the typical cerebral symptoms by a considerable period, especially in those cases which commence with tabetic symptoms.

*Paralyses of the extrinsic ocular muscles* occur about half as frequently as in tabes, and have exactly the same characteristics, the IIIrd nerve being most frequently involved.

**Cerebral Syphilis** is the term usually applied to relatively early, direct syphilitic disease of the brain and meninges. Its manifestations differ very materially from those of the parasyphilitic diseases, and the ocular symptoms are of special diagnostic importance. Cerebral syphilis is due essentially

to gummatous inflammation of the meninges and the walls of the cerebral blood vessels.

The chief form of brain syphilis is basal gummatous meningitis. It usually arises from the subarachnoid tissue in the region of the chiasma and spreads thence over the base of the brain. The optic nerves, chiasma, and tracts are generally involved. Papillitis, papillœdema, or post-neuritic atrophy are frequently found (about 13 per cent. each), and are usually bilateral. Visual defects are very common, and consist of amblyopia, not infrequently amaurosis, and defects in the fields of vision. Of the latter many cases show homonymous hemianopia from affection of one tract, fewer cases temporal hemianopia. Central scotoma and other signs of retrobulbar neuritis also occur. The IIIrd nerve is paralysed in a third of the cases, less commonly the Vth and VIth, and least frequently the IVth. The IIIrd and VIth are often affected on both sides. The trigeminal paralysis is always unilateral and often causes neuroparalytic keratitis. Pupillary changes occur, dependent upon the IIIrd nerve lesions. In many cases the process is limited to a small area, oculomotor paralysis, or an affection of the visual path being the only signs except headache. A very characteristic feature of basal gummatous meningitis is the inconstancy and variability of the symptoms, temporary and recurrent visual and oculomotor disturbances being very common.

Isolated gummata may give rise to the signs of cerebral tumour, complicated by the fact that they are often multiple. Syphilitic disease of the cerebral vessels is responsible for a large proportion of cases of thrombosis, hæmorrhage, softening, &c.

**Intracranial Tumours.** The commonest ocular manifestation of intracranial tumours is papillœdema (*vide* p. 387).

Analysis of 200 cases of intracranial tumour treated at the National Hospital, Queen Square, shows the following results (Paton) :—

(1) Precentral tumours are nearly always associated with papillœdema fairly severe in character. (2) Postcentral tumours are nearly always associated with papillœdema, as a rule moderate, and often of very short duration. (3) Temporosphenoidal tumours are always associated with papillœdema of about the same degree of severity as in frontal tumours. (4) Of subcortical tumours about one-half develop papillœdema —as a rule, moderate in degree—and, as in the case of parietal tumours, frequently of short duration. (5) Optic thalamus and

mid-brain tumours are almost invariably associated with papillœdema of very great severity. (6) Cerebellar tumours are constantly accompanied by papillœdema of a grave character. (7) Extra-cerebellar tumours, as a rule, develop papillœdema of a grave character. (8) Of pontine tumours, only about one-half develop papillœdema, and then only when neighbouring parts of the brain, especially the cerebellum, have become involved: the papillœdema when it does develop is usually very severe. (9) Ventricular tumours develop a moderate papillœdema.

There are two regions of the brain, the pons and the central white matter of the cerebral hemispheres, in which tumours frequently develop without causing papillœdema. Some cases of meningeal tumours in which the brain substance escapes do not develop papillœdema. When a tumour directly or indirectly exercises pressure on the chiasma or optic nerves atrophy may occur without preceding papillœdema (*vide* pp. 389, 393). In these cases loss of vision may precede ophthalmoscopic signs, and may first be manifest as a unilateral central scotoma (*vide* pp. 393, 396, 411).

Homonymous hemianopia is due in about half the cases in which this symptom is present to tumours of the occipital lobes. About 20 per cent. are due to involvement of one tract, either direct or as a pressure symptom. Relatively few are due to involvement of the internal capsule or external geniculate body. Heteronymous hemianopia is much rarer and is due to pressure on the chiasma and tracts by tumours of the pituitary body or distension of the third ventricle.

Paralysis of ocular muscles is relatively rare and nearly always a distant pressure symptom. One or both VIth nerves are often affected, the IIIrd nerve rarely, the IVth practically never. Conjugate lateral deviation of the eyes, which is common in cerebral hæmorrhage and to a less extent in cerebral softening, is rare with tumours of the cerebrum. It is more frequent with cerebellar tumours. Paralyses as true localising symptoms may of course occur with tumours of the crus, pons, &c. To this category belongs trigeminal paralysis, with or without neuroparalytic keratitis: it is rare with cerebral, commoner with cerebellar tumours.

**Intracranial Abscess.** Cerebral abscess occurs about three times as often as cerebellar. The majority of cerebral abscesses are due to middle ear disease and affect the temporal lobes. Others are due to traumatism and generally affect the parietal lobes. Rarer causes are metastatic infection, usually derived from the

lungs, frontal sinus empyema, and orbital cellulitis. A still greater proportion of cerebellar abscesses is due to otitis media.

Nearly half the cases have papillœdema: it is not infrequently on the side of the abscess only, and in bilateral cases the swelling is generally greater on this side. This sign has therefore greater localising value in intracranial abscess than in tumour. Papillœdema persists longer after operation for abscess than for tumour, or may even only then commence. As might be expected, optic atrophy is rare during the acute stage: its presence militates against the diagnosis of abscess. Ophthalmoscopic changes are rarer with extradural abscesses.

Homonymous hemianopia indicates a lesion of the occipital lobe, which is rarely due to otitis.

Partial unilateral IIIrd nerve paralysis is fairly common, and the combination of unilateral ptosis and mydriasis has almost pathognomonic significance of ipsilateral cerebral or cerebellar abscess. Partial IIIrd nerve paralysis with contralateral hemiplegia points to abscess of the temporal lobe with pressure on the IIIrd nerve and internal capsule, or more rarely to implication of the cerebral peduncle. Paralysis of the VIth nerve is not common, but is found rather oftener in cerebellar than cerebral abscess: it is generally ipsilateral, but has little localising value. Paralysis of the Vth nerve is rare. Nystagmus is very common with cerebellar abscess, but rare with cerebral. In otitic cases it may be due to disease of the labyrinth.

**Intracranial Aneurysm and Sub-arachnoid Hæmorrhage.** Intracranial aneurysms are not very rare, and may rupture spontaneously or after head injury into the subarachnoid space. Spontaneous cases are often preceded by severe headaches on stooping or exertion and tinnitus. The rupture is usually accompanied by sudden very acute headache, vomiting and dizziness. Coma may rapidly supervene. Meningeal irritation is shown by stiffness of the neck and often by Kernig's sign. The ocular signs are ocular palsies, especially of the IIIrd or VIth nerve; moderate papillœdema; retinal hæmorrhages, usually multiple in the neighbourhood of the disc, rather large, and often subhyaloid; vitreous hæmorrhage; proptosis; and defects in the visual fields. There is always blood in the cerebrospinal fluid, as shown by lumbar puncture.

**Acrocephaly** (*Syn.—Oxycephaly*) is due to precocious union of certain cranial sutures: occipito-parietal and fronto-parietal

(*turricephaly, tower skull*), sagittal (*scaphocephaly*). Asynchronous fusion of bones leads to a lop-sided skull (*plagiocephaly*). The great wing of the sphenoid is displaced so that the orbit becomes shallow, causing more or less proptosis. In the early stages there is papillœdema, but more commonly only the later stage of post-neuritic optic atrophy is seen. The amount of atrophy varies in degree. The papillœdema is probably due to increased intra-cranial pressure, owing to continued growth of the brain in a restricted space. Divergent strabismus, horizontal nystagmus and mental deficiency are common. Most of the patients are males. Acrocephaly may be associated with syndactylism (Apert's disease).

**Encephalitis.** Ocular palsies usually usher in an attack of *encephalitis lethargica*. Ptosis is the commonest feature, and other branches of the IIIrd nerve are specially involved. The muscles are usually only partially paralysed, and generally recover. Diplopia is an early symptom, and nystagmus may be present. Papillœdema is rare and the pupils are usually normal. The general symptoms are lethargy, with great muscular debility, and other signs of an acute general infection. The disease is often followed by Parkinsonian tremor (paralysis agitans), and in the later stages spasmodic conjugate deviation of the eyes occurs (oculogyric crises) accompanied by synergic movements of the head and neck. Oculogyric crises are relieved by benzedrine (up to 30 mg. a day).

Acute *polioencephalitis* accounts for not infrequent cases of paralytic squint following a febrile attack in young children. The VIth nerve is most often involved.

**Meningitis.** In *tuberculous meningitis* a moderate degree of papillitis is common (about 25 per cent.) and is generally bilateral. Papillœdema occasionally occurs and indicates the combination of solitary with miliary tubercle. Tubercle in the choroid is frequent and of great diagnostic importance. A review of the literature tends to show that it is less common in tuberculous meningitis than in generalised miliary tubercle, but my own observations lead me to think that it is much commoner than is generally thought. It is often found only a day or two before death. There are often partial ocular pareses, usually of the IIIrd nerve, especially in the form of ptosis. Bilateral IIIrd paralysis is almost unknown, a point of distinction from syphilitic basal meningitis. Unilateral partial VIth nerve paralysis also occurs. Not infrequently

there is a kinetic (not paralytic) conjugate deviation of the eyes and head to one side.

In *epidemic cerebro-spinal meningitis* papillitis is frequently present, never papillœdema ; it is due to a descending infective neuritis. In the early stages there is often kinetic strabismus or conjugate lateral deviation of the eyes. A characteristic sign is the widely open palpebral aperture, often associated with very infrequent blinking. Paralysis of the VIth nerve, usually unilateral, is commoner than that of the IIIrd, though divergent strabismus due to the latter cause has been frequently noted. Total IIIrd nerve paralysis is rare (cf. Gummatous Basal Meningitis). The pupils vary much, usually showing miosis in the early stages, mydriasis when coma sets in : loss of reaction to light is relatively rare. Conjunctivitis and keratitis sometimes occur, and many cases of metastatic endophthalmitis (*q.v.*) in children are due to the Weichselbaum meningococcus, though it is a relatively rare complication of the disease.

Still and others have shown that the sporadic acute basal meningitis of children is due to the meningococcus. A peculiarity of this disease which I have frequently seen at Great Ormond Street Children's Hospital is complete amaurosis with normal fundi and normal pupil reactions, pointing to the action of toxins on the higher visual centres. The blindness may persist for many weeks after subsidence of other symptoms, and sight may be completely restored. Chronic basal meningitis sometimes shows the same feature, but in these cases optic neuritis and post-neuritic atrophy may occur from secondary hydrocephalus and pressure of the distended third ventricle upon the chiasma and tracts.

*Purulent meningitis* occurs occasionally in typhoid, and more rarely in pneumonia, influenza, scarlet fever, measles, and septicæmia. In typhoid the diagnosis is difficult, but the presence of papillitis and ocular paralyses points in this direction. Metastatic purulent meningitis, with papillitis or retrobulbar neuritis, occurs in children from obscure causes. Middle ear disease is a not uncommon cause of purulent meningitis. In this condition papillitis or papillœdema is usually due to complications, such as sinus thrombosis or cerebral abscess. When ocular paralysis occurs, the VIth nerve is usually affected, rarely the IIIrd (cf. Intracranial Abscess). The facial nerve is most frequently involved, the paralysis often causing lagophthalmia. Conjugate deviation of the eyes is not uncommon. Metastatic endophthalmitis is rarer than in

epidemic cerebro-spinal meningitis. The diagnosis of otogenous meningitis from tuberculous may be difficult or impossible.

**Hydrocephalus.** In the congenital and the early acquired hydrocephalus of infancy optic atrophy is not infrequently found. Papillœdema occurs only rarely in spite of the increased intracranial pressure. This fact is doubtless due to the relief of pressure by the enlargement of the skull and the resiliency of the fontanelles and gaping sutures, as well as to the very gradual development. The eyeballs usually deviate downwards, and upward movements are much restricted. This is sometimes due to bulging of the thin orbital plate of the frontal bone, which may be even absorbed. Not infrequently there is considerable proptosis.

The acquired hydrocephalus of later life, after the fontanelles and sutures have closed, can often only be diagnosed with certainty by encephalography. The cardinal signs of increased intracranial pressure—headache, vomiting and papillœdema—are present, and to these is often added ataxia of the cerebellar type. The cases are often diagnosed as intracranial tumours, in which localising signs are not infrequently absent or masked. Bitemporal hemianopia may give a clue to the true ætiology, being due to pressure on the chiasma and tracts by the bulging floor of the third ventricle. In some cases there is evidence of previous meningitis, but the most characteristic feature is often the variability of the symptoms. Remissions and intermissions of long duration occur, and recovery or arrest of the condition is not uncommon, often, however, with defective vision due to post-neuritic atrophy.

**Fractures of the base of the Skull.** Unilateral facial paralysis is the commonest cranial nerve lesion in fractures of the base of the skull (22 per cent. of cases): the VIth (4 per cent.), IIIrd (2 per cent.), Vth (1·6 per cent.), and IVth (1 per cent.) follow in order of frequency. Fractures of the base from falls upon the head, &c., sometimes pass through the optic foramen and involve the roof of the orbit: occasionally both optic foramina are broken. It may happen that the nerve is directly injured or compressed by hæmorrhage; more frequently, however, owing to the intimate union between the dura mater and periosteum, the optic nerve is injured indirectly, probably by laceration of the small meningeal vessels feeding it. If the injury is severe, in two to four weeks signs of primary optic atrophy appear and progress to total atrophy. Papillœdema indicates hæmorrhage into the nerve sheath, and may occur from basal hæmorrhage without

fracture of the optic foramen. These injuries may cause concentric contraction of the field of vision, or quadrant and other sectorial defects: central scotoma appears to be rare. Most cases with rapidly developing papillœdema die. Pigmentation in and around the disc may follow hæmorrhage into the sheath. The pupil reactions vary and are not pathognomonic, but there is usually mydriasis on the side of the lesion.

**Statistics.** The statistics on this page (in percentages), derived from Uhthoff's very extensive investigations of the literature and of cases in the Breslau clinic, give some idea of the frequency of

| | Papillœdema | Papillitis | Optic Atrophy. | Homonymous Hemianopia | Bitemporal Hemianopia. | III Paralysis. | IV Paralysis. | VI Paralysis. | V Paralysis. | Conjugate Deviation of the Eyes. | Nystagmus. |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Cerebral Tumour | 53 | 18 | 8 | 17 | 1 | 14 | 0·6 | 11 | 6 | 3 | 4 |
| Cerebellar Tumour | 53 | 24 | 11 | — | — | 5 | 2 | 18 | 12 | 1·5 | 25 |
| Cerebral Abscess | 23 | 21 | 0·3 | 9 | — | 19 | 1·6 | 10 | 4 | 6 | 4 |
| Cerebellar Abscess | 23 | 22 | — | — | — | 14 | — | 12 | 4 | 6 | 42 |
| Cerebral Syphilis | 14 | 12 | 14 | 11 | 6 | 34 | 5 | 16 | 14 | 1 | 8 |
| Cerebral Hæmorrhage | 11 | 6·5 | 1 | 29 | — | 9 | — | 8·4 | 1 | 28 | 10 |
| Cerebral Softening | 1·4 | 2·2 | 0·8 | 40 | — | 2·4 | — | 0·3 | 2 | 12 | 1·6 |
| Tuberculous Meningitis | 5 | 29 | 1 | — | — | 18 | 1 | 12 | 5 | 8 | 10 |
| Internal Hydrocephalus | 23 | 20 | 19 | — | — | 6 | — | 13 | — | 1 | 13 |
| Pituitary Body Tumours | 9 | 8·5 | 21 | 3 | 32 | 17 | 2·5 | 6 | — | — | 4 |
| Fractures of the Base of the Skull | 9 | | 4 | — | — | 2 | 1 | 4 | 1·6 | 2·5 | 3 |

important physical signs in certain diseases of the nervous system already discussed. Too much reliance must not be placed upon the percentages, culled as the cases are from very various sources.

## CHAPTER XXX

### Ocular Manifestations of other Diseases

THE most important ocular manifestations of other diseases than those of the central nervous system have already been discussed incidentally, and it will suffice here merely to enumerate them.

**Infectious Diseases.** Mucopurulent conjunctivitis and corneal ulcers are the chief ocular complications of *measles*. They are rare in *scarlet fever*, as is also albuminuric retinitis. Corneal ulcers are common in *small-pox*. *Vaccinia* of the eyelids is not uncommon, and may affect the cornea, usually secondarily, sometimes causing disciform keratitis. *Diphtheria* sometimes attacks the conjunctiva ; it may cause cycloplegia (*vide* p. 539), and rarely paralysis of the external rectus. *Erysipelas* may cause abscesses and gangrene of the lids, orbital cellulitis, and thrombosis of the orbital veins and cavernous sinus. Optic neuritis occurs in *typhoid fever*. Conjunctivitis and the herpetic types of keratitis are common in *influenza*, which also causes iritis and optic neuritis.

**Diseases of the Respiratory Tract.** Conjunctival hæmorrhages are common in *whooping cough*, and retinal hæmorrhages may also occur. Herpes corneæ occurs in *pneumonia*, but it is remarkable that hypopyon ulcer is rare. Apical *phthisis* may cause irritation of the sympathetic fibres, leading to dilatation of the ipsilateral pupil.

**Diseases of the Circulatory System.** Pulsation of the retinal vessels, embolism and thrombosis of the central artery, arterio-sclerosis, and thrombosis of the central vein of the retina have already been sufficiently discussed. Aneurysm at the root of the neck may cause dilatation of the pupil on the same side. In congenital heart disease the retinal vessels are usually dark and greatly engorged ; or the veins alone may be abnormally large ; retinal hæmorrhages are not uncommon.

**Diseases of the Blood.** *Chlorosis* has often been held responsible for papillitis, but it is extremely doubtful if the diagnosis is accurate. Retinal hæmorrhages, sometimes accompanied by white spots of exudate, occur in the *secondary anæmias* of carcinoma, ankylostomiasis, &c., and are a pro-

minent sign in *pernicious anæmia*, which really belongs to this group. In this disease they are often of characteristic colour, as in leukæmia (*vide* p. 371). The ophthalmoscopic signs of *leukæmia* have already been described. Hæmorrhages in and about the eyes are common in scurvy and purpura; rare in hæmophilia. Great *loss of blood* leads to amblyopia or amaurosis, and may be followed by bilateral optic atrophy (*vide* p. 404). Severe ocular symptoms very rarely follow traumatic hæmorrhage, as in war injuries, but most commonly result from intestinal or uterine hæmorrhage. Though both eyes are usually affected there is often an interval of days between them. The discs are hazy at first, later becoming atrophic, with constricted vessels. In some cases slight improvement of sight eventually occurs.

**Diseases of the Organs of Digestion.** *Oral sepsis*, especially pyorrhœa alveolaris, is an undoubted cause of iridocyclitis (*vide* p. 274), and probably causes choroiditis and other forms of subacute or acute endophthalmitis. Infective lesions in the mouth may spread by continuity, especially along the veins of the pterygoid plexus, setting up orbital cellulitis or thrombosis of the cavernous sinus. Lamellar cataract is associated with hypoplasia of the enamel of certain teeth. The lacrymal gland is not infrequently affected in *parotitis*, which may be associated with irido-cyclitis (*uveo-parotid inflammation*) (*vide* p. 277). Symmetrical inflammation of the lacrymal and salivary glands is characteristic of *Mikulicz' disease* (*vide* p. 650). Absorption of bacterial toxins from the intestinal canal is almost certainly a cause of iridocyclitis and other obscure inflammations of the uveal tract. Night blindness is associated with some *diseases of the liver*, *e.g.*, cirrhosis, and jaundice causes yellow discoloration of the conjunctiva, but yellow vision (xanthopsia) is much less common than has been thought.

Malignant nasopharyngeal growths form 0·4 per cent. of all cases of cancer; 38 per cent. of cases show ophthalmo-neurological symptoms, these being the earliest sign in 16 per cent. of cases (Godtfredsen). The Vth and VIth nerves are most frequently involved; more rarely the IIIrd, IVth and the optic nerve. Quadrantic and hemianopic lesions are rare, thus distinguishing these cases from lesions in the neighbourhood of the sella Turcica. The presence of abducens paralysis, especially if associated with impairment of vision, or Horner's syndrome, or exophthalmos or enlargement of cervical glands, should suggest a nasopharyngeal growth. The treatment is purely radiological.

**Diseases of the Kidneys.** Renal retinopathy and uræmic amaurosis have already been discussed.

**Metabolic Diseases.** Ocular complications are common in *diabetes mellitus*, but bear little relation to the severity of the disease; they occur chiefly in long-standing cases, the most frequent being diabetic cataract, retinitis, intraocular hæmorrhages, and retrobulbar neuritis. Œdema of the pigment epithelium on the back of the iris is often seen in microscopical specimens, but iritis is seldom met with. Remarkable changes in the refraction of the eye, both in the direction of hypermetropia and myopia, not infrequently occur in diabetics, due to alterations in the refractive index of the cortex of the lens, probably brought about by osmotic changes (*vide* p. 327). Paralyses of both extrinsic and intrinsic ocular muscles also occur. *Gout* has been held responsible for deposits in the conjunctiva (concretions), conjunctivitis, marginal ulcers of the cornea, episcleritis, scleritis, iritis, and other conditions: it is indirectly the cause of ocular lesions through the kidneys and vascular system. *Rheumatism* is an indefinite entity. Acute rheumatism is doubtless an infective disease: it practically never gives rise to iritis, but may cause embolism of the central artery of the retina indirectly by its effects on the cardiac valves. It rarely causes optic neuritis. Chronic rheumatism is also probably due to organisms or bacterial toxins, and is thus responsible for iritis, cyclitis, episcleritis, and retrobulbar neuritis. Interstitial keratitis has been described in *myxœdema:* over-feeding with thyroid may cause cataract and amblyopia.

**Diseases of the Generative Organs.** It can scarcely be doubted that the profound changes which the generative organs, especially in the female, undergo at puberty, in menstruation, parturition, and the climacteric, are often associated with disorders of metabolism and other pathological conditions. Of these the albuminuric retinitis of pregnancy is the most impeccable example among ocular complications. Loss of vision, starting in retrobulbar neuritis and followed by papillœdema and peripapillary retinal hæmorrhage, occurs in severe cases of hyperemesis gravidarum, probably as part of the syndrome of Wernicke's encephalopathy. It may be due to some vitamin deficiency, *e.g.*, $B_1$. Many other ocular complications have been described, *e.g.*, conjunctivitis associated with the menses, but their relationship to diseases of the generative organs is obscure. The diseases due to gonorrhœa and syphilis have been described elsewhere.

# SECTION VII

## DISEASES OF THE ADNEXA OF THE EYE

### CHAPTER XXXI

### Diseases of the Lids

**Anatomy.** The lids are covered anteriorly by skin and posteriorly by mucous membrane—conjunctiva tarsi; they end in a free edge about 3 mm. broad—margo intermarginalis. The substance of the lids consists of muscle, glands, blood

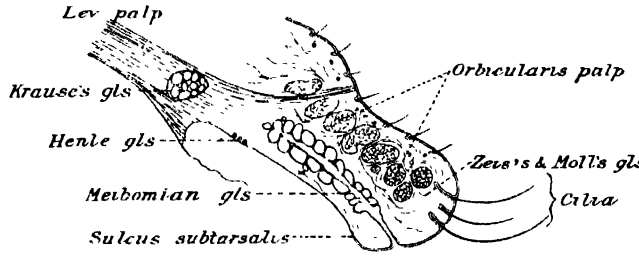



FIG. 326.—Diagram of sagittal section of upper lid.

vessels, and nerves, all bound together by connective tissue, which is particularly dense at the posterior part, where it forms a stiff plate—the tarsus (Fig. 326).

The skin of the lids differs from that of the rest of the body merely in its thinness, its loose attachment, and the absence of fat in its corium. It is covered with fine downy hairs, which are provided with small sebaceous glands, and there are also small sweat glands. At the anterior border the hairs are specially differentiated to form a protection to the eyeball. The cilia or eyelashes are strong, short, curved hairs, arranged in two or more closely set rows. Their sebaceous follicles, like the cilia themselves, are specially differentiated, and are called *Zeis's glands*. Apart from being larger, they are identical with other sebaceous glands. The sweat glands near

the edge are also unusually large and are known as *Moll's glands*. They are situated immediately behind the hair follicles, and their ducts open into the ducts of Zeis's glands or into the hair follicles, not direct on to the surface of the skin as elsewhere.

The margin or free edge of the lid is the part between the anterior and posterior borders—the intermarginal strip or margo intermarginalis (Fig. 327). It is covered with stratified epithelium, which forms a transition between the skin and the conjunctiva proper. The anterior border is rounded; the posterior, which lies in contact with the globe, is sharp. The capillarity induced by this sharp angle of contact is of importance in the proper moistening of the surface of the eye. Immediately anterior to the posterior border is a single row of minute orifices, just visible to the naked eye. These are the orifices of the ducts of the Meibomian glands. Between this

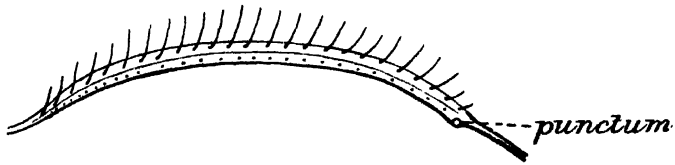



FIG. 327.—Diagram of intermarginal strip.

row of puncta and the anterior border is a fine grey line, which is important in operations in which the lid is split, as it indicates the position of the loose fibrous tissue between the orbicularis palpebrarum and the tarsus.

The tarsus consists of dense fibrous tissue; it contains no cartilage cells, so that the term tarsal cartilage is only justified in so far as it defines the consistence of the plate. Imbedded in the tarsus are some enormously developed sebaceous glands, the *Meibomian glands*. They consist of nearly straight tubes, directed vertically, each opening by a single duct on the margin of the lid. The tubes are closed at the upper end, and have numerous small cæcal appendages projecting from the sides, filled with fatty glandular epithelium. The glands number from twenty to thirty, being rather fewer in the lower than in the upper lid.

The large bundles of the orbicularis palpebrarum occupy the space between the tarsus and the skin. The main central band of the levator palpebræ superioris is inserted into the upper border of the tarsus; an anterior slip passes between

the bundles of the orbicularis to be inserted into the skin of the middle of the lid; a posterior slip is inserted into the conjunctiva at the fornix. The inferior rectus and oblique muscles send fibrous strands forwards into the lower lid to be attached to the tarsus and palpebral ligament.

Besides these striped muscles there is a layer of unstriped muscle in each lid. These constitute the superior and inferior tarsal muscles of Müller. The fibres of the former arise among the striped fibres of the levator, pass down behind it, and are inserted into the upper border of the tarsus. The inferior lies below the inferior rectus and is inserted into the lower tarsus.

The arteries of the upper lid form two main arches, superior and inferior, the former lying between the upper border of the tarsus and the orbicularis, the latter in a similar position just above the hair follicles. In the lower lid there is usually only one arch near the free edge. There are two venous plexuses in each lid: a post-tarsal passing into the ophthalmic veins, and a pre-tarsal opening into subcutaneous veins.

The sensory nerve supply is derived from the trigeminal. The third nerve supplies the levator palpebræ, the seventh the orbicularis, and the sympathetic Müller's muscles.

## Inflammation of the Lids

Almost any of the inflammatory conditions which affect the *skin* in general may attack the lids. Erysipelas is dangerous in that it may spread to the orbit, leading to cellulitis and atrophy of the optic nerve, thrombosis of the cavernous sinus, or meningitis. Herpes ophthalmicus is often mistaken for erysipelas; its unilaterality, strict localisation to the course of branches of the ophthalmic nerve, and the characteristic formation of vesicles should prevent this mistake; permanent scarring remains after the attack. Eczema of the lids is common, especially associated with phlyctenular conjunctivitis (*q.v.*) in children, and with atropine irritation (*q.v.*). Dermatitis is not uncommonly caused by cosmetics, *e.g.*, such as contain orris root and volatile oils, and especially by applications for dyeing the lashes. Abscesses, boils, anthrax pustule, and ulcers of various kinds may affect the skin of the lids. Œdema of the lids may be inflammatory or passive. It is often associated with chemosis of the conjunctiva in severe conjunctivitis. Great œdema is often caused by bites of parasites, gnats, &c., and by styes, abscesses, and chancre of the lid. In unilateral œdema the condition of the lacrymal

sac and nasal duct should be investigated; it is often due to lacrymal abscess. Situated above the internal palpebral ligament it suggests empyema of the frontal sinus; in the lower lid empyema of the antrum. Localised œdema may be due to periostitis of the orbital margin. In all cases of œdema the condition of the eyeball must be determined, with the assistance of Desmarres' retractors if necessary. Œdema of the lids may be caused by serious purulent inflammation of the globe (panophthalmitis), of Tenon's capsule, by phlegmon of the orbit or thrombosis of the cavernous sinus. Passive œdema may be due to nephritis, heart disease, &c., or it may be angioneurotic. Chronic thickening of the lids, resembling œdema, but harder in consistency, may follow recurrent attacks of erysipelas—so-called *solid œdema*.

Blepharitis is a chronic inflammation of the margins of the lids. It may manifest itself as a simple hyperæmia, differing from that caused by weeping, exposure to tobacco smoke, and so on, in being more persistent. The causes and treatment are the same as for the more severe forms of blepharitis. True blepharitis occurs in two forms. In *squamous blepharitis* small white scales, like dandruff, accumulate among the lashes; the latter fall out readily, but are replaced without distortion. If the scales are removed the underlying surface is found to be hyperæmic, but not ulcerated. The condition is probably a seborrhœa.

In *ulcerative blepharitis* yellow crusts glue the lashes together; on removing them small ulcers, which bleed easily, are seen around the bases of the lashes. The lashes fall out or are easily pulled out, and often are not replaced, or grow in a distorted form, owing to injury to the follicles. Blepharitis causes redness of the edges of the lids, itching, soreness, lacrymation, and "photophobia."

The sequelæ of the ulcerative form are serious. If not treated energetically and with perseverance the disease is extremely chronic, causing or being accompanied by chronic conjunctivitis. Care must be taken to distinguish true blepharitis from matting together of the lids by conjunctival discharge; in the latter case removal of the crusts reveals quite normal lid margins. The ulceration is liable to extend deeply, so that the hair follicles are destroyed. Only a few small, scattered, distorted cilia are then found (*madarosis*).

When the ulcers heal the cicatricial tissue contracts. Neighbouring hair follicles are drawn out of place, and a false direction is given to the remaining cilia, so that they may rub

against the cornea (*trichiasis*). Or the development of cicatricial tissue may be extreme, so that the edge of the lid becomes hypertrophied and droops in consequence of its weight (*tylosis*).

The lower lid is particularly liable to be displaced by prolonged ulcerative blepharitis. The contraction of the scar tissue drags the conjunctiva over the margin; the posterior lip of the intermarginal strip, instead of being acute-angled, becomes rounded, so that its capillarity is impaired (*vide* p. 618). Tears then tend to run over (*epiphora*), a condition which is accentuated if the punctum becomes everted, so that it ceases to lie in accurate contact with the bulbar conjunctiva (*vide* p. 650). The continual wetting of the skin with tears leads to eczema, which is followed by contraction. The condition is made worse by perpetually wiping the eyes, so that eventually *ectropion* is developed. This causes still more epiphora, a vicious circle being set up.

The causes of blepharitis are multitudinous. The patients are usually children debilitated from living under poor hygienic conditions, or from disease, *e.g* anæmia, tubercle, syphilis, measles, &c. The condition may follow chronic conjunctivitis, or be induced by the same causes, especially smoky atmosphere, heat (stokers, cooks), late hours, &c. It may result from a neglected diplobacillary blepharo-conjunctivitis. It is undoubtedly often associated with uncorrected errors of refraction, especially hypermetropia and astigmatism, which probably act by inducing reflex hyperæmia. Occasionally parasites cause blepharitis, *e.g.*, blepharitis acarica, due to demodex folliculorum, and phthiriasis palpebrarum, due to the pediculus pubis, very rarely to pediculus capitis. In the latter condition the cilia are covered with black nits, an appearance being produced which is easily recognised when once seen.

*Treatment.* The local treatment of blepharitis must be energetic in the ulcerative form. The crusts must first be removed. This is effected most easily by soap and water, followed by thorough bathing with hot borax or bicarbonate of soda lotion, 3 per cent. The application softens the deposits, so that they can be picked or rubbed off with a pledget of cotton-wool. When the crusts have been entirely removed the surface is covered with penicillin ointment, provided the organism involved is sensitive to this drug. If it is not, a sulphonamide preparation may be used (albucid ointment, 30 per cent.). When the infection has been eliminated stimu-

lating applications may be applied—an ointment of yellow oxide of mercury, ammoniated mercury, or ichthyol (5 per cent.), which is gently well rubbed in for at least five minutes, so as to insinuate it into the hair follicles. These procedures should be repeated three times a day. In most cases, if the treatment is carried out properly, there is a speedy cure. Unfortunately the treatment is seldom carried out satisfactorily. It is useless merely to smear ointment on the surface of the crusts. It must be applied to the inflamed tissues and rubbed well into the lashes. The treatment should be continued for 2 or 3 weeks after apparent cure, as organisms lie hidden in the follicles and the inflammation is likely to recur.

In more severe cases, when organisms insensitive to penicillin are present, or when the above treatment is improperly done, other methods may be applied. Thus protargol, 15 to 20 per cent., may be rubbed into the margins of the lids with a stump camel's hair brush until a lather is formed ; this usually takes five minutes. Or the surface may be thoroughly cleaned with bicarbonate lotion, and silver nitrate, 2 per cent., painted on. Daily application of a solution composed of equal parts of 0·5 per cent. solution of crystal violet and brilliant green in equal parts of alcohol and water, or a similar ointment (ung. tinctorium) has cured some cases. All loose lashes should be pulled out with epilation forceps. If diplobacilli are present a zinc lotion should be used.

Attention must be directed to the hygienic surroundings, and to the general health. Errors of refraction must be corrected.

**Syphilis.** A primary sore is occasionally found on the lid margins, commencing in the conjunctiva. It may be caused by a kiss or by removing a foreign body with the tongue. There is generally a small ulcer, covered with scanty greyish secretion and much indurated about the base. If situated near the outer canthus the pre-auricular gland is enlarged, if near the inner canthus the submaxillary, in accordance with the distribution of the lymphatic vessels. The swelling of the glands is always suggestive of syphilis or tubercle, but in all doubtful cases scrapings should be examined for spirochætes and the blood examined by the Wassermann test. General penicillin treatment or other energetic constitutional measures should at once be undertaken (*vide* p. 696).

Gummata occur in the lids sometimes, and occasionally may cause enormous thickening of the tarsus (*syphilitic tarsitis*). Isolated gumma may be mistaken for a chalazion. In syphilitic tarsitis the lid may be so swollen and hard that

it is impossible to evert it. The pre-auricular gland is swollen. If the onset is slow there is little pain ; sometimes the swelling is rapid and very painful. Gummata usually respond rapidly to appropriate anti-syphilitic treatment with penicillin or even mercury and iodides.

**Vaccinia.** The margin of the lid is occasionally inoculated from the recently vaccinated arm of a baby. Often the inoculated margin in turn inoculates the opposing margin of the other lid. Usually the pustule is at the outer canthus, and the pre-auricular gland is swollen and painful. The history generally serves to elucidate the case. Sometimes

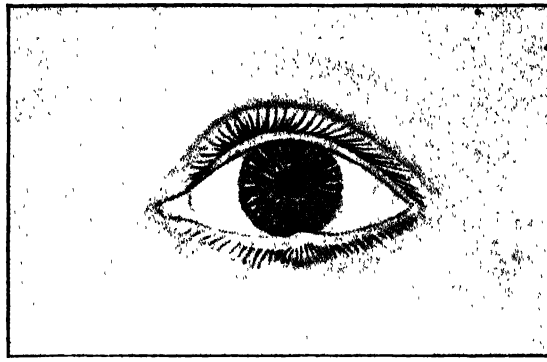


FIG. 328.—Hordeolum.

the cornea becomes affected, a keratitis resembling disciform keratitis resulting, *i.e.*, a grey disc, denser at the margin.

### INFLAMMATION OF THE GLANDS OF THE LIDS

Hordeolum or stye is a suppurative inflammation of one of Zeis's glands (Fig. 328). In the early stages the gland becomes swollen, hard and painful, and usually the whole edge of the lid is œdematous. An abscess forms which generally points near the base of one of the cilia.

The pain is considerable until the pus is evacuated. Styes often occur in crops, or may alternate with boils on the neck, carbuncles, or acne. Like these conditions, the disease shows deficient resistance of the body to the invasion of staphylococci. It is commonest in young adults, but may occur at all ages, especially in debilitated persons. Not infrequently it will be found that faulty drains account for the defective health.

*Treatment.* Hot compresses should be used in the early stages. When the abscess points it may often be evacuated by pulling out the corresponding cilium; but this is usually effected more satisfactorily by an incision with a small knife. It should be remembered that such an incision is very painful unless novocain or novutox has been injected. The pus should be thoroughly squeezed out and a hot compress applied.

If crops of styes occur the general health must receive attention. When associated with boils or carbuncles, the urine should be tested for sugar, especially in adults. A general course of penicillin treatment in addition to the local application of the ointment usually clears up the infection.

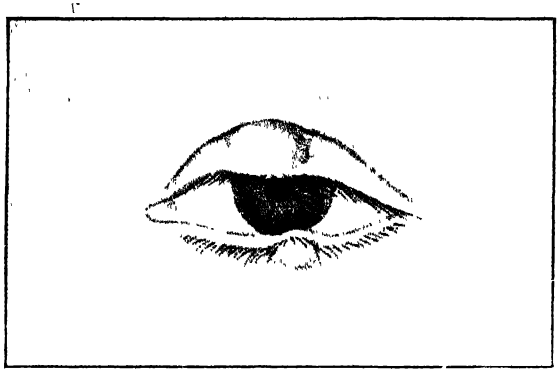


FIG. 329.—Chalazion.

In penicillin resistant or obstinate cases a staphylococcic vaccine, preferably autogenous, should be used.

Constipation must be counteracted, and tonics are useful, especially iron in some form.

**Hordeolum internum** is comparatively rare. It is a suppurative inflammation of a Meibomian gland of exactly the same type as the hordeolum externum or stye. It is often called a suppurating chalazion, and some may be due to secondary infection of a chalazion. The inflammatory symptoms are more violent than in external stye, for the gland is larger and is imbedded in dense fibrous tissue. The pus appears as a yellow spot shining through the conjunctiva when the lid is everted. It may burst through the duct or through the conjunctiva, rarely through the skin.

*Treatment* is the same as for external type, except that the incision should be made exactly as for a chalazion (*vide infra*).

**Chalazion** (*Syns.*—*Tarsal Cyst, Meibomian Cyst*) is a chronic inflammatory affection of a Meibomian gland. The gland tissue becomes replaced by granulation tissue containing giant cells; the disease is not caused by the tubercle bacillus, but is probably caused by the chronic irritation of an organism of low virulence. The gland becomes swollen, increasing in size very gradually and without inflammatory symptoms. Patients usually seek advice on account of the disfigurement (Fig. 329). The smaller chalazia are difficult to see, but are readily appreciated by passing the finger over the skin. If the lid is everted the conjunctiva is red or purple over the nodule, in later stages often grey, or rarely, if infection has occurred (*vide* Hordeolum internum), yellow. The grey appearance is due to alteration in the granulation tissue. This is not very vascular at any stage, but in the later stages the vessels retrogress, the nourishment of the tissue fails and it becomes converted into a jelly-like mass. Only under such conditions is the term "cyst" really applicable. Complete spontaneous resolution very rarely occurs. The contents may be extruded through the conjunctiva, and in these cases a fungating mass of granulation tissue often sprouts through the opening, keeping up conjunctival discharge and irritation. Sometimes the granulation tissue is formed only in the duct of the gland, from which it projects as a reddish-grey, somewhat translucent nodule on the intermarginal strip (*marginal chalazion*).

Chalazia are often multiple or occur in crops. They are commoner in adults than in children.

*Treatment.* Quite small chalazia may be left alone: it is very difficult to evacuate them satisfactorily by the ordinary method. Larger chalazia must be incised and


FIG. 330. — Beer's knife (devised for cataract extraction: now used only for lid operations).

thoroughly scraped. The conjunctival sac is well anæsthetised with 2 per cent. pantocain and a drop of adrenaline instilled. The lid is then everted and the site of the chalazion carefully examined. At the point of greatest discoloration a few crystals (not many) of solid cocaine are placed upon the surface and allowed to dissolve, or ½ c.c. of 4 per cent. novocain solution may be injected subcutaneously in the lid. A vertical incision is then made though the palpebral conjunctiva with a sharp scapel or Beer's knife (Fig. 330). Any semi-fluid contents which may be present escape. A small sharp spoon (Fig. 331) is then inserted into the orifice and the walls of the cavity are thoroughly scraped. The bleeding soon stops, and no dressing is usually necessary. A simple boric acid lotion is ordered for a few days.

The patient should be warned that the swelling will remain for a while. This is due to the resistant walls, formed by the fibrous tissue of the tarsus; the cavity is thus kept dilated and becomes filled with blood. Sometimes, especially if the scraping has not been sufficient, granulation tissue sprouts from the wound. This must be snipped off with scissors, curved on the flat, after application of pantocain; the cavity should be again scraped out.

Fig. 331.—Sharp spoon.

Very hard chalazia are occasionally met with, particularly near the canthi; it is possible that some of these are true adenomata of the glands. They may require excision, since it may be impossible to scrape them out efficiently.

If a marginal chalazion is not treated the granulation tissue protrudes from the mouth of the gland and may organise into a greyish, somewhat translucent lump of fibrous tissue on the lid margin. It is disfiguring and rather difficult to remove without leaving an irregularity in the line of the lid. It is best treated by diathermy, with a small needle as the active electrode. A current of 200—300 milliampères is passed for one second, and the operation repeated if necessary.

## Anomalies of Position of the Lids

**Trichiasis** (*θρίξ, τρίχος*, a hair) is the condition of distortion of the cilia, so that they are directed backwards and rub

against the cornea (*vide* p. 180). A few only of the lashes may be affected, or the condition may be due to entropion involving the whole margin of the lid. It may also be caused by congenital distichiasis (*vide* p. 646).

The symptoms are those of a foreign body continually present in the eye—irritation, pain, conjunctival congestion, reflex blepharospasm, lacrymation. Superficial opacities and vascularisation of the cornea are produced; recurrent ulcers of the cornea are not infrequently due to this cause.

Any condition causing entropion (*q.v.*) will cause trichiasis, trachoma and spastic entropion being among the most common. Other causes are blepharitis, and the scars resulting from injuries, burns, operations, diphtheria, &c.

*Treatment.* Isolated misdirected cilia may be removed by epilation, which must, however, be repeated every few weeks. A better mode of treatment is to destroy the hair follicle by diathermy or electrolysis. With the former a fine needle is inserted into the hair follicle and a current of 150 milliampères is applied for one to two seconds. With the latter the flat positive pole is applied to the temple; the negative, a fine steel needle, is introduced into the hair follicle: a current of two milliampères is used. The negative pole is determined by placing the terminals in saline, when bubbles of hydrogen are given off by it. The strength of current can be gauged by the rate of evolution of gas. It should be remembered that electrolysis is extremely painful and tedious; the pain may be avoided by injecting novocain into the margin of the lid. If the current is of the proper strength, the bubbles evolved at the site of puncture cause the formation of a slight foam, and the lash with its bulbous root can be easily lifted out.

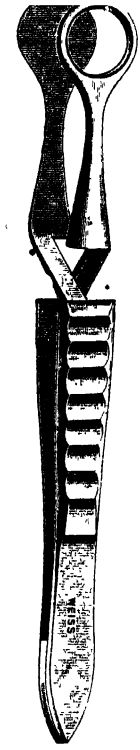


FIG. 332.—Chalazion clamp; useful for holding the lid everted in incising chalazia. The ring is placed on the conjunctival surface and surrounds the chalazion.

If many cilia are displaced, operative procedures must be resorted to. Since they are nearly allied to those performed

for entropion, which is generally present, they will be described later (*vide* p. 628).

**Entropion** (ἐν, in, τρέπειν, to turn), rolling in of the lid, occurs in two forms, spastic and cicatricial. The symptoms are those of the trichiasis (*q.v.*) which is induced.

*Spastic entropion* is due to spasm of the orbicularis. Strong contraction of the circularly arranged fibres tends not only to approximate the lid margins, but also to turn them inwards or outwards, according to the mechanical support afforded by the globe and orbital contents. If the support is insufficient, entropion is produced. This is well seen when the eyeball has been removed, but it also occurs when the globe is deeply set owing to absence of orbital fat, &c., especially if the skin of

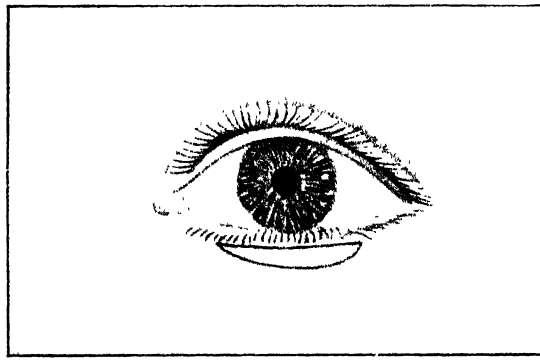


FIG. 333.—Diagram of skin and muscle operation.

the lids is also redundant. These conditions are found *par excellence* in old people, who are therefore very liable to spastic entropion. It is also caused by tight bandaging, and is favoured by narrowness of the palpebral aperture (blepharophimosis). Spastic entropion is almost invariably restricted to the lower lid.

*Cicatricial entropion* is caused by cicatricial contraction of the palpebral conjunctiva; in the worst forms, found in trachoma, the tarsal plate is also bent and distorted, sometimes by atrophic, sometimes by hyperplastic, changes. It is an exaggeration of the effect produced by the various causes of trichiasis (*q.v.*).

*Treatment of Spastic Entropion.* If due to bandaging, the condition is often cured by simply leaving off the bandage. Wearing an artificial eye relieves the symptoms when the

eyeball has been removed. In the spastic entropion of old people temporary relief may be obtained by placing a roll of lint or plaster horizontally just above the margin of the orbit, and bandaging it firmly in position; or the lid may be slightly everted by painting collodion on the skin or by pulling it out with a strip of adhesive plaster. Injection of 1 c.c. of 80 per cent. alcohol subcutaneously along the edge of the lid, with or without canthoplasty, has been advocated (Weekers).

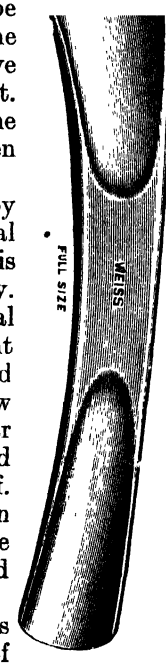



FIG. 334. Lid spatula, which should be made of metal.

Permanent relief can be obtained only by operation. The simplest method is the removal of a strip of skin and muscle. Pantocain is instilled and novocain injected subcutaneously. An oval area of skin, with the long axis horizontal and varying in width according to the amount of entropion and of superfluous skin, is marked out with a scalpel or Beer's knife just below the site of greatest displacement. The upper incision must be close to the margin of the lid (Fig. 334). The piece of skin is dissected off. The underlying fibres of the orbicularis are then dissected off with forceps and knife, until the tarsus is exposed. Two or three sutures should be inserted.

Wheeler's operation (Figs. 335, 336) is less likely to be followed by recurrence. A strip of orbicularis 4 mm. wide is drawn upwards and outwards over the malar bone and secured to the periosteum by catgut sutures.

In cases of spastic entropion with much blepharospasm, canthoplasty is sometimes indicated. It consists in widening the palpebral aperture by dividing the outer

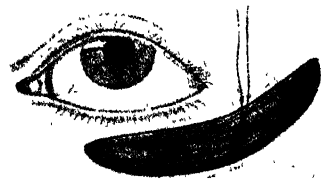


FIG. 335.

canthus. The lids are separated with the fingers in such a manner as to put the canthus on the stretch. One blade of strong blunt-pointed scissors is introduced as far as possible

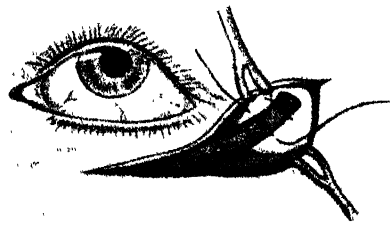


FIG. 336.

into the conjunctival sac behind the commissure. The entire thickness, including skin and conjunctiva, is divided horizontally by a single cut. If only a temporary effect is required, no sutures are inserted. If it is desired permanently to enlarge the palpebral aperture, the conjunctiva is sutured to the skin. Temporary canthoplasty is sometimes indicated in

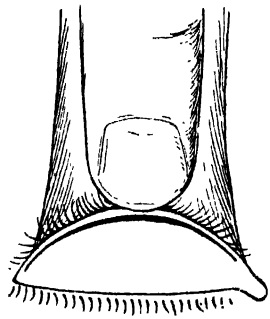


FIG. 337.—Diagram of modified Burow's operation for entropion.

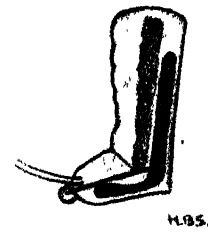


FIG. 338. — Diagram of modified Burow's operation for entropion.

other conditions than spastic entropion, *e.g.*, in simple severe blepharospasm, such as occurs in phlyctenular conjunctivitis, in acute purulent conjunctivitis with much swelling of the lids, and in removal of an enlarged eyeball or an orbital tumour.

*Treatment of Cicatricial Entropion.* Many plastic operations have been devised for the relief of cicatricial entropion: only the more simple will be described here. The principles

governing the various operations are : (1) altering the direction of the lashes, (2) transplanting the lashes, (3) straightening the distorted tarsus. Subcutaneous injection of novocain or a

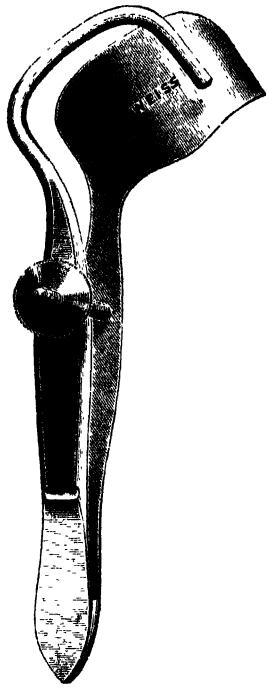


Fig. 339.—Desmarres' entropion forceps for right eye.

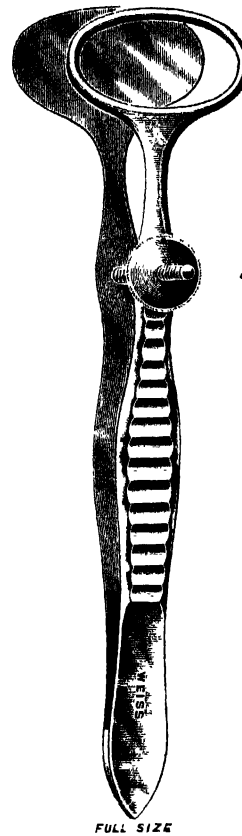



Fig. 340.—Wilde's entropion forceps.

general anæsthetic is indicated ; the former method does not obviate all pain, especially if the tarsus is cut.

The simplest procedure is some modification of Burow's operation. The lid is everted over the end of a metal lid spatula (Fig. 334). A horizontal incision through the conjunctiva and passing completely through the tarsal plate, but not through the skin, is made along the whole length

of the lid in the sulcus subtarsalis, *i.e.*, about 2—3 mm. above the posterior border of the intermarginal strip (Fig. 337). Care must be taken not to wound the punctum or canaliculus. The temporal end of the strip may then be divided by a vertical incision through the free edge of the lid, including the whole thickness. In this manner the edge of the lid is left attached only by skin, and when cicatrisation has occurred the edge is turned slightly outwards, so that the lashes are directed away from the eye. Relapses are not uncommon, however, and the operation may have to be repeated. The edge of the lid may be kept everted during the process of

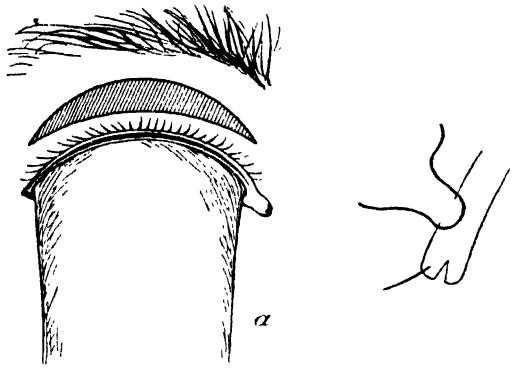



FIG. 341.—Diagram of Jaesche-Arlt operation for entropion.

healing by means of a spindle-shaped pad of oiled silk. The pad is kept in position by sutures suitably applied.

Fig. 338 illustrates an alternative operation. The incision is made as before. The tarsal plate is pared down to a chisel edge along the whole length and mattress sutures are passed through the plate and lid margin, emerging through the grey line (p. 618); they are tied over glass beads, thus bending the lid margin forwards and upwards.

In the Jaesche-Arlt operation the zone of hair follicles is transplanted to a slightly higher position. The lid is split from the outer canthus to just outside the punctum along the grey line (*vide* p. 618) between the lashes and the orifices of the Meibomian glands. During this procedure the globe is protected by the spatula inserted between it and the lid, or held by a lid clamp (Figs. 339, 340). The incision extends between the tarsus and the orbicularis for a depth of 3—4 mm., so that the zone containing the hair follicles is thoroughly loosened

(Fig. 341). A crescentic piece of skin is then removed from the lid. The lower incision extends through the skin down to the tarsus at a distance of 3—4 mm. from the edge of the lid and parallel with it for its whole length. The middle part of the upper incision is 6—8 mm. from the edge of the lid. The crescentic piece of skin thus marked out is removed, without taking any orbicularis. The two skin incisions are then sutured. In this manner the zone of lashes is transplanted to a higher level. The gaping wound in the intermarginal strip may be filled in with a graft of mucous membrane; this tends to prevent the follicles from being drawn down again when the wound cicatrises. Care should be taken not to produce ectropion by removing too much skin.

**Ectropion,** rolling out of the lid, occurs in several forms, the chief being spastic, cicatricial, senile, and paralytic. The symptoms are due to the epiphora induced and to the chronic conjunctivitis caused by exposure. In severe cases the cornea may suffer from imperfect closure of the lids.

*Spastic ectropion* results from blepharospasm when the lids are well supported by the globe and when they are short, firm, and without redundant skin. It is therefore seen in children and young patients, and is readily induced by phlyctenular conjunctivitis (*vide* p. 169). Mechanical ectropion is caused by extreme proptosis or thickening of the conjunctiva, such as occurs after purulent conjunctivitis and trachoma. In the latter disease the tarsus is often distorted. Upper and lower lids are frequently affected simultaneously.

*Cicatricial ectropion* results from destruction of the skin by injury, burns, ulcers, gangrene, operations, &c. Caries of the orbital bones is a common cause in children. Chronic conjunctivitis and blepharitis also cause cicatricial ectropion, which is increased by the wetting of the skin with tears and the eczema thereby induced.

*Senile ectropion* is found only in the lower lid, and is due to relaxation of the tissues and degeneration of the orbicular muscle fibres. The condition is increased by the conjunctivitis and epiphora which are set up.

*Paralytic ectropion* results from the laxity of the lids induced by paralysis of the orbicularis. Only the lower lid is affected, the upper being kept in contact with the globe by its own weight.

In long-standing cases of ectropion the exposed conjunctiva becomes dry and thickened, red and very unsightly.

*Treatment.* Non-operative treatment is chiefly serviceable

in spastic ectropion. Here a well-fitting bandage, unless contra-indicated by other factors, will often cure the displacement. In ectropion paralyticum, the condition is cured only by restoration of the innervation. The slighter degrees of senile ectropion are also amenable to non-operative treatment, though it may be advisable to slit the canaliculus in order to cure epiphora (*vide* p. 651). The patient should be instructed not to pull the lid down when wiping the eye.

A large variety of operations has been devised for ectropion: only the simpler procedures will be described.

Snellen's sutures are indicated in some cases of spastic and senile ectropion. In this operation two loops of thread, inserted at the junction of the middle with the inner and outer thirds of the lid, through the ectropionised conjunctiva, are

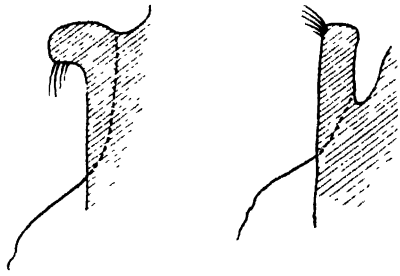


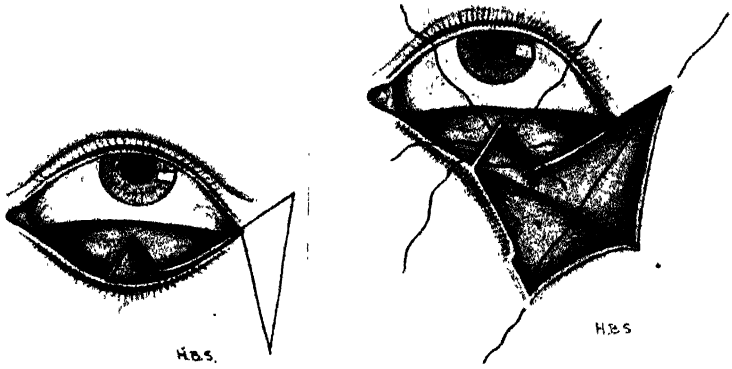
FIG. 342.—Diagram of Snellen's sutures for ectropion.

made to hold the fornix in its proper position (Fig. 342). The sutures are then tied over pieces of rubber tubing laid vertically, and may be tightened from day to day so as to cause the formation of cicatricial bands along their tracks: this was more effectual in the days when antiseptic precautions were not observed. The effect is seldom permanent.

In paralytic ectropion lateral tarsorrhaphy may be indicated. In this operation the palpebral aperture is shortened by uniting the lids at the outer canthus. The edges of the upper and lower lids are freshened for the requisite distance, the lashes being excised. The lids are then sutured together as in central tarsorrhaphy (*vide* p. 227).

In many cases of ectropion, especially senile, the lower lid is stretched and elongated. The ectropion may then be cured by shortening the lid as in Dimmer's modification of Kuhnt's operation (Figs. 343, 344). A triangular piece of conjunctiva

and tarsus is excised, the apex of the triangle being towards the fornix. The lid is then split along the grey line from the triangle to the outer canthus. A triangular area of skin is



FIGS. 343, 344.—Dimmer's modification of Kuhnt's operation for ectropion.

removed at the outer canthus and the skin is slid outwards so that the gap in the tarsal plate is closed, the requisite length of the margin of the lid at the outer canthus being

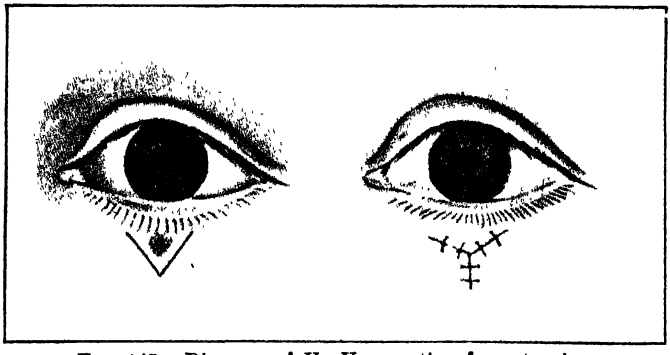


FIG. 345.—Diagram of V—Y operation for ectropion.

denuded of cilia. Care should be taken to carry the upper skin incision up and out, so that the lid will be drawn slightly upwards. It is quite as effectual to remove the triangle of tarsus at the outer canthus, and this avoids the necessity of splitting the lid.

In most of these operations restoration of the normal position is facilitated by dissecting off the strip of thickened conjunctiva at the margin of the lid.

In the slighter cases of cicatricial ectropion the V—Y operation of Wharton Jones is indicated (Fig. 345). A V-shaped incision, with the apex away from the lid margin, is made through the skin, the limbs of the V enclosing the cicatrix. The skin is freed from the underlying tissues and is also well undermined at the edges. The margins of the incisions are sutured in such a manner that a Y-shaped cicatrix results: the edge of the lid is thus raised to its normal position.

More extensive cicatricial displacement requires some form of blepharoplastic operation, employing skin grafts (Wolfe or Thiersch) or flaps of skin taken from the cheek or temporal region, or strips of fascia lata. Each such case must be treated on its own merits and will often exercise the ingenuity of the surgeon.

FIG. 346.—Symblepharon.

**Symblepharon** (*συν*, with, together, *βλέφαρον*, eyelid) is the condition of adhesion of the lid to the globe (Fig. 346). Any cause which produces raw surfaces upon two opposed spots of the palpebral and bulbar conjunctiva will lead to adhesion if the spots are allowed to remain in contact during the process of healing. Such causes are burns from heat or caustics, ulcers, diphtheria, operations, &c. Bands of fibrous tissue are thus formed, stretching between the lid and the globe, involving the cornea if this has also been injured. The bands may be narrow, but are more frequently broad, and may extend into the fornix so that the lid is completely adherent to the eyeball over a considerable area (symblepharon posterior). Bands limited to the anterior parts and not involving the fornix are called symblepharon anterior. Total symblepharon, in which the lids are completely adherent to the globe, is rare.

Pronounced adhesions cause impairment of mobility of the eye, so that diplopia may be complained of. The adhesion

may be so intimate that it is impossible to close the lids efficiently, lagophthalmia, with its baneful consequences, resulting. There is often much disfigurement.

*Treatment.* The prevention of symblepharon is of the utmost importance (*vide* p. 436). When it is already established operation is necessary. Symblepharon anterior is usually easily remedied by dividing the bands and preventing reformation of adhesions in the manner already described. When the bands are broad, and especially if there is symblepharon posterior, the separation of the lid from the globe is difficult. There is no guide to the limitations of sclerotic and tarsus, and great care has to be exercised lest the globe be punctured. The prevention of re-formation of adhesions is much more difficult, and is successful only if the raw surfaces are covered with conjunctival or mucous membrane grafts (*vide* p. 436).

**Ankyloblepharon** (*ἀγκύλη*, a thong, *βλέφαρον*, eyelid) is adhesion of the margins of the two lids. It may be either a congenital condition or due to burns, &c. It may be partial or complete, and is often combined with symblepharon. The treatment depends upon the amount of symblepharon. If it is very extensive operation may be contraindicated. In other cases the lids are separated and kept apart during the healing process. If the adhesion extends to the angle of the lids, the latter must be covered with an epithelial graft, otherwise the condition will recur.

**Blepharophimosis** (*βλέφαρον*, eyelid, *φιμός*, a muzzle) is the condition in which the palpebral fissure appears to be contracted at the outer canthus. The outer angle is really normal, but is obscured by a vertical fold of skin. The latter is due to eczematous contraction of the skin following prolonged epiphora and blepharospasm. Mere narrowing of the palpebral aperture is often called blepharophimosis, and may be a congenital condition: it is really a form of ankyloblepharon.

The condition may require no treatment, disappearing spontaneously after the inflammation has subsided. In other cases canthoplasty is indicated.

**Lagophthalmia** (*λαγώς*, a hare) is the condition of incomplete closure of the palpebral aperture when the eyes are shut. It may be due to narrowing of the lids from cicatrisation or congenital deformity, ectropion, paralysis of the orbicularis, proptosis due to exophthalmic goître, orbital tumour, &c., or to laxity of the tissues and absence of reflex blinking in people who are extremely ill or moribund. Owing to exposure the

cornea becomes epidermoid (xerosis corneæ) or keratitis sets in. The treatment is that of keratitis e lagophthalmo (*q.v.*).

**Ptosis** (πίπτειν, to fall) is the term given to drooping of the upper lid, due to paralysis or defective development of the levator palpebræ superioris. Ptosis may also be caused by thickening and increased weight of the lid (*vide* p. 621). The condition may be unilateral or bilateral, partial or complete. In the higher degrees the lid hangs down, covering the pupil more or less completely and interfering with vision. An attempt is made to counteract the effect by overaction of the frontalis and by throwing back the head, the eyes being pulled downwards by the inferior recti. A very characteristic attitude is thus adopted. Forced contraction of the frontalis causes the eyebrows to be raised and throws the skin of the forehead into wrinkles. Partial ptosis may be masked by this means, but becomes manifest if the patient is asked to look up while the eyebrows are fixed by firm pressure with the fingers against the bone.

Ptosis may be congenital or acquired. The congenital form is usually, but not invariably, bilateral, and is due in most cases to defective development of the muscles. Some cases have been proved to be caused by maldevelopment of the third nucleus. The condition is not infrequently hereditary. There is nearly always defect in the upward movement of the eyes, due partly to absence of the posterior insertion of the levator into the fornix (*vide* p. 619), partly to coincident maldevelopment or defective innervation of the superior rectus. It may be pointed out here that defective upward movement of the eyes is the commonest congenital defect of bilaterally associated extrinsic muscles.

Acquired ptosis is usually unilateral. It may be part of the symptom-complex of paresis or paralysis of the whole of the third nerve, or may be due to paresis or paralysis of the branch supplying the levator. Isolated ptosis without other signs of oculomotor paralysis may result from disease of upper level centres (cerebral ptosis). Acquired ptosis may also be due to direct injury of the muscle or its nerve supply, as by wounds, fractures, &c. Mechanical ptosis is due to deformity and increased weight of the lid brought about by trachoma, tumours, &c.; it also occurs from lack of support in phthisis bulbi, anophthalmia, &c. Bilateral ptosis may occur in the acquired form, notably as part of the syndrome of myasthenia gravis.

The amount of ptosis sometimes alters with the position of

the globe, attaining its highest pitch in abduction of the eye, its least in adduction or attempted adduction. Occasionally in both the congenital and acquired forms the lid rises when the jaw is moved, as in mastication, though it remains immobile when an attempt is made to look upwards (*vide* p. 566). This is an example of synkinesis or associated movement.

*Treatment.* In cases of paralysis of the third nerve treatment must be directed to removal of the cause. The fact that this nerve is so frequently affected in syphilis must be borne in mind; these cases respond to treatment better than others. In cases of incurable paralysis and in congenital and mechanical ptosis the deformity can be removed only by operation. In

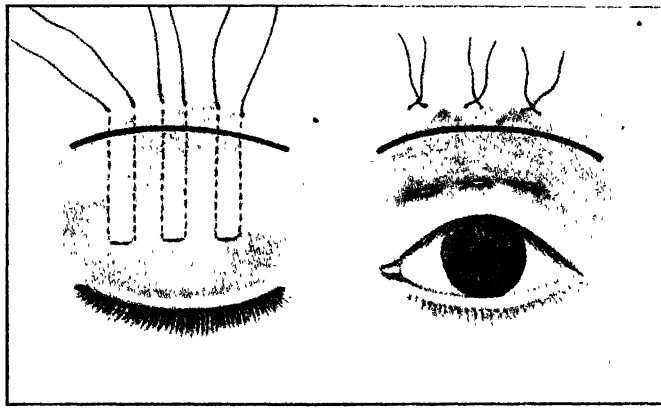


FIG. 347.—Diagram of Hess's operation for ptosis.

complete paralysis of the third nerve operation is usually contraindicated on account of the abduction of the eye. If the lid is raised in these cases the diplopia becomes manifest; simultaneous advancement of the internal rectus may diminish the diplopia and the deformity, but is unsatisfactory and unlikely to give a permanent result.

Operations for ptosis ameliorate the condition but seldom give permanent results. In slight cases excision of an elliptical area of skin, with or without excision of the underlying fibres of the orbicularis, improves the appearance temporarily.

Of the many operations which have been devised for the more severe cases Hess's operation is one of the simplest. The eyebrow is shaved. An incision is made in the line of the eyebrow for 2·5 cm. The skin of the lid is then undermined through this

incision so that it is completely separated from the orbicularis and tarsus over its whole area. Three silk sutures are then inserted as shown in Fig. 347 ; or the lid may be raised by narrow strips of fascia lata similarly inserted.

A better but more difficult operation is Greeves's modification of Motais'. A controlling suture is first inserted in the conjunctiva, immediately above the cornea, and the eye depressed as far as possible by its means. The superior rectus tendon and its attachment to the globe are then exposed by a horizontal incision through the conjunctiva and a silk thread is then passed

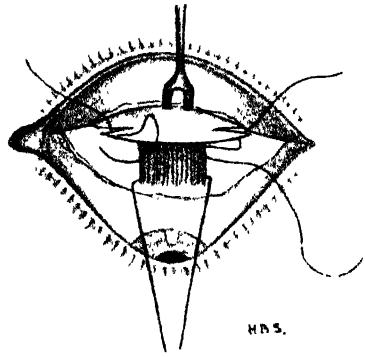


FIG. 348.—Greeves's modification of Motais' operation : showing the controlling suture, and the exposed rectus tendon.

under the tendon ; the two ends of the thread are secured by Spencer Wells forceps.

Next the upper lid is everted and the conjunctiva above the incision seized by forceps and dissected upwards until the upper edge of the tarsal plate is exposed.

The upper edge of the tarsal plate appears as a convex rounded border ; this is gripped centrally by catch-forceps, on each side of which a thin strip of tarsus is cut with a fine pair of bent scissors, from without inwards and within outwards respectively, each strip being left attached centrally (Fig. 348), the width of the uncut area of tarsal plate between the strips being about the same as that of the superior rectus tendon : as long a strip of tarsal tissue as possible should be aimed at. Sutures are then passed through the ends of the strips and again through the corresponding edge of the superior rectus tendon (Fig. 349). The sutures are drawn tight without being tied, in order that a judgment may

be made of the relative positions of the eye and eyelid; the position of the edge of the lid should be such that it slightly overlaps the upper part of the cornea. If the position of the lid is judged not to be correct, those parts of the sutures which have been passed through the tendon should be withdrawn and re-inserted in the tendon in a suitable position, either higher up or lower down as required.

After healing has taken place and all reaction has disappeared, it will frequently be found that when the eyeball is raised, the skin of the upper lid is apt to fall in an unsightly fold over the lashes; the skin is flabby and does not seem to possess a normal tone. A horizontal strip of skin of suitable width is removed from

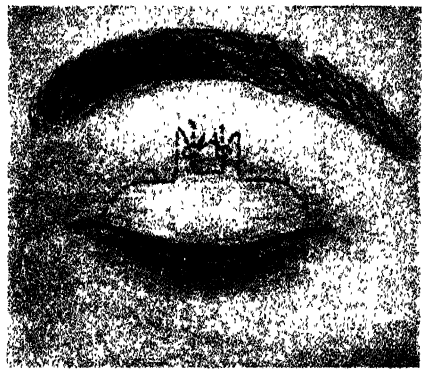


FIG. 349.—The dotted lines indicate the position of the tarsal plate, tarsal strips, superior rectus tendon, and sutures, when the operation is completed. (Greeves.)

the upper lid, the position of the lower skin incision corresponding roughly to that of the upper edge of the tarsal plate. The sutures which join the edges of the skin incision are carried through the deep tissues in such a way as to stretch the skin over the tarsus and to produce a fold in the skin of the eyelid in the normal position.

### INJURIES OF THE LIDS

Injuries of the most various kinds—contusions, wounds, burns, &c.—are very common. They must be treated upon general principles, but special attention must be directed to three points—(1) wounds of the skin of the lids, (2) injury of the bones of the orbit, (3) injury of the eyeball.

**Wounds** in the direction of the fibres of the orbicularis gape

little and heal without conspicuous scarring; hence surgical wounds should be made in this direction as far as possible. Vertical wounds gape, cause disfiguring cicatrices, and often lead to ectropion, or other distortion, especially if there is adhesion to the subjacent bone. The worst wounds are such as sever the lid vertically in its whole thickness. If they do not unite by first intention a notch (traumatic coloboma) is left in the lid margin, and disfigurement, lagophthalmia, and epiphora result. Vertical wounds severing the canaliculus require special care (*vide infra*).

**Injury to the bones of the orbit** may affect the orbital margin or deeper parts. Fractures involving the margin may be diagnosed by careful palpation—unevenness, crepitation, &c. Fractures of the walls of the orbit often manifest themselves by *emphysema*. It is due to communication of the subcutaneous tissues with the nasal air sinuses, air being forced into the tissues on blowing the nose, sneezing, straining, or coughing. There is great swelling, with a peculiar soft crepitation on palpation. Fracture of the orbital bones may be followed by retraction of the globe (traumatic enophthalmos), or may be part of a more serious fracture of the base of the skull. In the latter event the optic foramen may be involved, causing laceration or compression of the optic nerve (*vide* p. 404).

**Injuries involving the globe** require special care both in diagnosis and treatment. In every case of injury of the lids the eyeball must be very carefully examined. Palpation, which should be very gentle, will usually demonstrate considerable reduction of intraocular pressure if the eye is injured; it indicates rupture of the globe. Inspection may be difficult on account of excessive swelling and ecchymosis. In such cases the eye must be examined at all costs, the lids being separated by retractors, under an anæsthetic if necessary.

**Contusions** are often more alarming in appearance than in reality. There is great swelling and ecchymosis both in the lids and conjunctiva. In all cases a guarded prognosis should be given, for it may be impossible to determine the full extent of the injury to the orbit (*vide* p. 670), or the eye (*vide* p. 437).

*Treatment.* Simple contusions with ecchymosis require only cold compresses: a simple boric lotion is ordered for cleansing the conjunctival sac, and boric ointment to prevent the lids from sticking together.

Emphysema should be treated with a pressure bandage, and all straining, blowing of the nose, and so on, must be avoided.

Wounds must be thoroughly cleansed with an antiseptic

lotion and brought together by sutures. On account of the rich blood-supply it is not necessary to make such a wide excision of the edges of a wound of the lid as it is elsewhere: only obviously contused and devitalised tissue should be excised. As a prophylactic against infection the wound should be dusted with sulphonamide or penicillin powder. In wounds involving the canaliculus the inner cut end must be searched for, and the canaliculus slit up (*vide* p. 651). If this is not done before cicatrisation has occurred epiphora will follow, and it will be extremely difficult to obtain entry into the canaliculus in order to slit it up. Lacerated wounds are likely to leave ugly scars and deformity of the lids: these must be treated by plastic operation. If suppuration occurs the abscess must be opened and treated on general surgical principles.

**Burns.** It is important to diagnose the degree of a burn. First degree burns require cleansing and the application of sterile saline and penicillin packs every three hours during the day. Second degree burns should be cleansed. vesicles opened, and dead epithelium removed. On no account should any coagulant such as tannic acid, tannafax, &c., be used on the lids. It makes them rigid and immobile, so that it is impossible to apply satisfactory treatment to the eye, and much distortion of the lids follows. In third degree burns, after thorough cleansing and removal of dead tissue, a Stent mould is taken of the denuded area and a Thiersch graft applied (*vide* p. 510). A temporary tarsorrhaphy, permitting some access to the conjunctival sac for treatment, is helpful, and may be released when risk of cicatricial ectropion is past. A Thiersch graft relieves the pain considerably. The dressing (*vide* p. 510) over the graft is not changed for five or more days. Cicatricial deformities resulting from burns are corrected by plastic operation.

## Tumours of the Lids

**Benign tumours** include xanthelasma, molluscum, warts, nævus, angioma, and other tumours common to the skin and cutaneous glands.

Small clear *cysts* frequently occur among the lashes in old people, due to the retention of secretion of Moll's glands. They disappear if the anterior wall is snipped off.

*Xanthelasma* (ξανθός, yellow, ἔλασμα, a plate) or xanthoma is a slightly raised yellow plaque, most commonly found in the upper and lower lids near the inner canthus, and often sym-

metrical in the two lids and on both sides. The plaques are most common in elderly women. They grow slowly, and only require treatment on account of the disfigurement produced. They are sometimes associated with diabetes and excessive formation of cholesterin. They may be removed after subcutaneous injection of novocain, or destroyed by trichloracetic acid, electrolysis or radium.

*Molluscum contagiosum* is a small white umbilicated tumour, generally multiple. A substance resembling sebum can be squeezed out of it. Each tumour should be squeezed out after incision and the interior touched with solid silver nitrate stick.

*Nævus* or mole, usually pigmented, may occur on the lids, generally affecting the margin and involving both skin and conjunctiva. Two are often symmetrically situated on the lids of the same eye, indicating their origin at a time when the lids were still united. The microscopical appearance is characteristic, consisting of "nævus cells," often arranged in an alveolar manner. The growths very rarely take on malignant proliferation. They may be removed by diathermy.

*Hæmangioma*, often also called nævus, occurs in two forms —telangiectasis and cavernous hæmangioma. The former are bright red or port-wine coloured spots composed of dilated capillaries. The latter consist of dilated and anastomosing venous spaces lying in the subcutaneous tissue having all the characteristics of erectile tissue; they are not infrequently strictly localised as if partially encapsuled. They appear bluish when seen through the skin and form a swelling which increases in size on crying, lowering the head, &c. Cavernous hæmangiomata are rarely seen in adults, partly due to the fact that they are generally treated in early life, but possibly due to spontaneous atrophy of the growth and thickening of the skin.

Hæmangioma often follows the distribution of the first and second divisions of the Vth nerve. It may be associated with hæmangioma of the choroid and buphthalmia, and also with hæmangioma of the occipital cortex, causing homonymous hemianopia. The intracranial masses may be revealed radiographically, since they often contain calcareous deposits.

Telangiectases may be excised if small, or treated with radium, though radium applied near the eye may cause irradiation cataract. Electrolysis or carbonic acid snow may also be used. Cavernous hæmangiomata may be excised, preferably from the conjunctival surface, if small. If larger they may be treated by electrolysis. It is a good plan to use electrolysis

for a time until the tumour is consolidated with fibrous tissue, and then to excise the mass.

*Lymphangioma* occurs rarely in the lids.

Symmetrical soft swellings above the inner canthus are sometimes seen in elderly people. They are due to prolapse of the orbital fat through an aperture in the fascial septum.

**Malignant Tumours** include carcinomata and sarcomata, the former being much the more common. Epitheliomata (squamous-celled carcinomata) show a preference for spots where the character of the epithelium changes; they therefore commence generally at the edges of the lids. The patients are elderly; the preauricular gland may be enlarged, or if the growth is near the inner canthus, the submaxillary lymphatic glands. Any of the glands of the lid may in rare instances undergo carcinomatous proliferation.

The commonest malignant epithelial growth is the so-called rodent ulcer (basal-celled carcinoma), which shows a predilection for the inner canthus. It commences as a small pimple which ulcerates. If the scab is removed it is found that the edges are raised and indurated. The ulcer spreads very slowly, the epithelial growth extending under the skin in all directions and penetrating deeply. The surrounding structures are gradually destroyed; lids, orbit, and bones are invaded. The growth is only locally malignant and probably originates in the accessory epithelial structures of the skin—hair follicles and glands. The lymphatic glands are not affected. Rodent ulcer rarely occurs before forty years of age, and the rate of growth is of the order of years.

Sarcoma is rare; it may be round or spindle-celled, pigmented or non-pigmented. Round-celled growths, variously described as lymphoma, lymphosarcoma, pseudo-leukæmic tumours, &c., sometimes affect both orbits and all four lids causing symmetrical proptosis. Occasionally the patients show blood changes as in leukæmia, but these are usually absent. The growth is slow but continuous, and the eyes are lost from lagophthalmia. The malignant growths springing from nævi are usually called malignant melanomata.

*Treatment.* Epithelioma and sarcoma must be thoroughly extirpated by diathermy at all costs, even if it involves excision of the globe or exenteration of the orbit. Rodent ulcer, if small, should be excised. If larger, so as not to be amenable to operative treatment without sacrificing a good eye, it may be treated with radium or X-rays for a time, provided there is no involvement of the bones. Considerable

improvement, and even cure, has been reported from this treatment, but there can be no doubt that the results may be seriously misleading. The skin surface may show a firm scar, while the growth continues to spread beneath the surface. In any case it is wise to excise the scar freely after radium treatment, and for many months to keep a careful watch for any recurrence. In the later stages extensive plastic operations may have to be performed to protect the eyeball. When this becomes impossible the eye must be excised and the morbid tissues freely removed.

### Congenital Abnormalities of the Lids

Symblepharon, ankyloblepharon, ectropion, entropion and trichiasis occur occasionally as congenital malformations. Ptosis is a fairly common congenital defect.

**Distichiasis** (δίς, double, στίχος, a row) is a rare condition in which there are two complete rows of cilia, often in all four lids. The posterior row occupies the position of the Meibomian glands, which are reduced to mere sebaceous glands performing the normal function of lubricating the hairs. It causes trouble by rubbing against the cornea.

**Coloboma of the lid** is a notch in the edge of the lid. The gap is usually situated to the inner side of the middle line, generally affecting the upper lid. Two or more defects may occur in the same lid. Sometimes a bridge of skin links the coloboma to the globe, or there is a dermoid astride the limbus at the site of the coloboma. There are often other congenital defects of the eye or other parts of the body, *e.g.*, coloboma of the iris, accessory auricles, &c. Some cases are due to incomplete closure of the fœtal facial cleft, others probably to pressure of amniotic bands. Occasionally there is a notch at the outer part of the lower lid, associated with maldevelopment of the malar bone.

**Cryptophthalmia** (κρυπτός, hidden) is a very rare condition in which there is total ankylo- and sym-blepharon, associated with abnormality of the eye and often of the orbit. The skin passes continuously from the brow over the eye to the cheek.

**Microblepharon** is the condition in which the lids are abnormally small. They may be absent—*ablepharon*. These conditions usually occur only in cases of microphthalmia, or congenitally small eyes. Microphthalmia may be associated with a congenital *orbito-palpebral* cyst, causing a swelling of the lower lid. The cyst is connected with the eyeball, contains retinal tissue in its lining, and is due to defective closure of the fœtal fissure—an extreme case of ectatic coloboma of the choroid (*q.v.*). The eye-

ball may be apparently absent (*congenital anophthalmia*), but there are always microscopic vestiges of ocular tissues.

**Epicanthus** is a semilunar fold of skin, situated above and sometimes covering the inner canthus. It is usually bilateral, the eyes are far apart, and the bridge of the nose is flat. It may disappear as the nose develops. It is normal in Mongolian races.

**Neurofibromatosis** (*Syns.—Elephantiasis neuromatodes, plexiform neuroma, von Recklinghausen's disease*) may affect the lids and orbit. In typical cases the temporal region is also affected. The swollen lid and temporal region form a characteristic picture. The hypertrophied nerves can be felt through the skin as hard cords or knobs. The nerve fibres are little changed, the hyperplasia affecting the endo- and peri-neurium. In several cases the ciliary nerves have been found affected, both in the orbit, associated with true glioma of the optic nerve, and inside the globe, which in many cases has been buphthalmic. Operative measures are seldom satisfactory. The choroid and ciliary body may be much thickened by layers of dense nucleated fibrous tissue, probably derived from the cells of the sheath of Schwann. Laminated ovoid bodies resembling Paccinian corpuscles also occur.

# CHAPTER XXXII

## Diseases of the Lacrymal Apparatus

**Anatomy and Physiology.** The lacrymal apparatus consists of the lacrymal glands and the lacrymal passages.

The *lacrymal glands* of each eye consist of the superior or orbital gland, the inferior or palpebral gland, and the accessory lacrymal glands or Krause's glands. All are serous acinous glands, scarcely distinguishable, microscopically, from serous salivary glands, with which they are morphologically identical. The superior gland, about the size of a small almond, is situated in the lacrymal fossa at the outer part of the orbital plate of the frontal bone. Ten or twelve *lacrymal ducts* pass from it to open upon the surface of the conjunctiva at the outer part of the upper fornix. The inferior gland consists of only one or two lobules situated upon the course of the ducts of the superior portion. It can be seen when the eye looks down and in after the upper lid has been everted. The accessory or Krause's glands are microscopic acini, lying below the surface between the fornix and the edge of the tarsus. There are about forty-two in the upper, six to eight in the lower, fornix. The ducts of numerous acini unite to form a larger duct which opens on to the fornix.

The lacrymal passages consist of the puncta lacrymalia, the canaliculi, the lacrymal sac, and the nasal duct (Fig. 350). The *puncta lacrymalia* lie near the posterior border of the free margin of the lid about 6 mm. from the inner canthus. Each lid has one punctum and one canaliculus. The punctum is situated upon a slight elevation, larger in elderly people, the papilla lacrymalis. As already mentioned, this is visible under normal circumstances only when the lid is slightly everted (*vide* p. 84). The *canaliculus* passes from the punctum to the lacrymal sac. It is first directed vertically for about 1 to 2 mm., then horizontally for 6 to 7 mm. The canaliculi usually open separately through the outer wall of the lacrymal sac. The *lacrymal sac* lies in the lacrymal fossa formed by the lacrymal bone. When distended it is about 15 mm. long vertically, and 5 to 6 mm. wide. The fundus extends slightly

above the level of the inner tarsal ligament. The sac is surrounded by fibres of the orbicularis. The lower end narrows as it opens into the nasal duct. The *nasal duct*, varying much in size (12 to 24 mm. long, 3 to 6 mm. in diameter), passes downwards and slightly outwards and backwards, bounded by the superior maxilla and inferior turbinate, to open at the anterior part of the outer wall of the inferior meatus of the

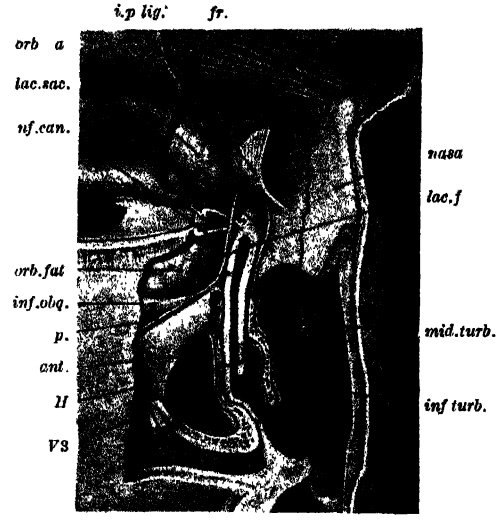



FIG. 350.—Canaliculi, lacrymal sac and nasal duct. *V3*, infraorbital nerve; *H*, valve of Hasner; *ant.*, antrum; *inf.obq.*, inferior oblique; *orb.fat*, orbital fat; *inf.can.*, inferior canaliculus; *lac.sac*, lacrymal sac; *i.p.lig.*, internal palpebral ligament (turned up); *fr.*, frontal process of superior maxilla; *nasal*, nasal bone; *lac.f.*, lacrymal fascia; *mid.turb.*, middle turbinate bone; *inf.turb.*, inferior turbinate bone; *p.*, periorbita. (Eugene Wolff. "Anatomy of the Eye and Orbit." Lewis, London.)

nose. The line of the duct is given by a point just outside the inner canthus and the groove between the ala of the nose and the cheek. The upper end of the nasal duct is the narrowest part. The canaliculi are lined by stratified epithelium, the lacrymal sac and nasal duct by columnar epithelium, lying upon a corium which contains a venous plexus. The mucous lining forms an imperfect valve at the orifice into the nose.

The *lacrymal secretion* is a slightly alkaline fluid containing sodium chloride as its chief constituent. The ordinary amount secreted is just sufficient to moisten the eyeball, and is lost

by evaporation. Only under reflex irritation, psychical or peripheral, is an excess secreted, and this is forced into the lacrymal sac and through the nasal duct into the nose during the act of winking, when the fibres of the orbicularis contract around the sac. It must be remembered that xerosis or dryness of the conjunctiva does not result from extirpation of the superior and inferior lacrymal glands, the moistening of the conjunctiva by Krause's glands and its own mucous cells being sufficient to prevent it. *Per contra*, epiphora does not result from extirpation of the lacrymal sac, except in the presence of psychical or peripheral stimuli to increased secretion. The tears have some slight antiseptic properties, owing to the presence of lysozyme.

### Diseases of the Lacrymal Gland

Diseases of the lacrymal gland are rare. *Dacryo-adenitis* occurs occasionally, usually going on to suppuration. Tubercle also occurs here. A permanent *fistula* may result from the bursting of an abscess in the gland. Spontaneous and traumatic *dislocation* of the gland have been described, a swelling being formed under the outer part of the upper lid.

*Dacryops* is a cystic swelling in the upper fornix, due to retention of secretion owing to blockage of one of the lacrymal ducts. It can only be distinguished from retention cysts of Krause's glands by its position.

*Tumours* of the lacrymal gland show a very marked resemblance to those of the parotid. In *Mikulicz' disease* there is symmetrical enlargement of the lacrymal and salivary glands, probably of a lymphomatous nature. Both parotid and lacrymal glands are enlarged in uveo-parotid inflammation (*vide* p. 277). Mixed tumours, in reality endotheliomata, containing cartilage, myxomatous material, &c., are the commonest form. Carcinomata and sarcomata are very rare.

All conditions which cause swelling of the gland may lead to impairment of movement of the eye. The globe is pushed downwards and inwards; movement outwards, and especially outwards and upwards, is limited. There may be some proptosis.

The rare diseases mentioned above must be treated on general principles.

### Diseases of the Lacrymal Passages

**Eversion of the lower punctum** occurs from laxity of the lids in old age, from chronic conjunctivitis, blepharitis, and any

cause leading to ectropion (*q.v.*). It causes epiphora, which in turn aggravates the condition (*vide* p. 633).

*Treatment.* In slight cases, especially in old people, the eversion may be sufficiently counteracted by making a small scar in the fornix just behind and below the position of the punctum. This is best done with the actual cautery, a fairly deep gutter being made. As the cicatricial tissue contracts the punctum is pulled inwards towards the eye.

If this fails the canaliculus should be slit up, the object being to bring the opened duct into apposition with the globe. It is therefore most important that the canaliculus should be slit up through its *posterior* wall.

The simplest method is the so-called "three-snip" operation. The vertical part is opened up, and then the horizontal part for 2 mm. This forms a triangular flap which is snipped off with scissors.

More extensive slitting of the lower canaliculus is performed as follows :—

Instruments required: Nettleship's dilator (Fig. 351), canaliculus knife (Fig. 352). The best form of canaliculus knife is the modification of Weber's in which the probe point is straight, not curved forwards as in the original instrument (Fig. 352). Pantocain is instilled into the conjunctival sac and novocain injected into the tissues around the canaliculus. The surgeon stands behind the patient. In operating upon the right side he everts the lower lid with his left thumb. With the right hand he inserts the point of Nettleship's dilator into the punctum, passing it directly downwards as far as it will go easily, then rotating it outwards and pushing it inwards along the canaliculus. In this manner the punctum is dilated. The knife is then taken and the probe point is passed into the punctum in the same manner, first downwards, then inwards. The back of the knife is directed forwards and slightly downwards. In this manner, as the knife is pushed inward, the posterior wall of the canaliculus is incised. While this manœuvre is being performed the lid is kept stretched out-


FIG. 351.—Nettleship's canaliculus dilator.

wards, so that the wall of the duct is kept taut against the edge of the knife. Care must be taken that the edge of the knife, which is directed towards the globe, does not injure the eye, though there is little danger of such an accident.

A probe should be passed along the incised canaliculus on the day following the operation, and occasionally on succeeding days, so as to prevent closure of the incision.

Under no circumstances should the canaliculus be slit up unless it is absolutely necessary. It should never be slit up *more* than is absolutely necessary.

FULL SIZE

FIG. 352.—Tweedy's canaliculus knife.

In some cases of eversion of the lower punctum a radical operation for ectropion may be necessary.

**Occlusion of the puncta** may be congenital, which is extremely rare, or cicatricial. Epiphora is caused. These cases are very difficult to treat. An endeavour should be made to slit up the occluded punctum—not the whole canaliculus. On inspection no trace of the punctum may be visible, but it is rare that some evidence of its presence cannot be seen on minute examination of the normal site with a loupe. The point of the dilator is inserted at this site, and may succeed in opening up the punctum sufficiently to admit the probe point of the canaliculus knife. There is usually no difficulty in knowing when the knife is in the duct, as it passes on in the proper direction quite easily. If this method fails to permit an entrance the canaliculus may be cut across vertically. When bleeding has stopped the inner cut end is examined with a loupe, and the probe point of the knife is inserted into it. If this also fails, and the upper punctum is patent, an attempt may be made to pass a small curved probe by the upper punctum into the sac and out into the lower canaliculus.

**Occlusion of the canaliculus** may be due to a scar (*vide* p. 642) or to a foreign body. Of the latter an eyelash is the commonest, less frequent a "concretion." An eyelash usually projects somewhat from the punctum, and is easily removed with forceps. Concretions are masses of the mycelium of a fungus, usually a streptothrix. They are removed by dilating the canaliculus and injecting 10 per cent. protargol.

**Congenital anomalies of the puncta and canaliculi** are

occasionally met with. The puncta may be absent or constricted; there may be two puncta in a lid, generally opening into the same canaliculus. Sometimes a groove is found instead of a canaliculus.

**Dacryocystitis** or inflammation of the lacrymal sac is not uncommon, especially among the lower classes. It is generally chronic. There is epiphora, aggravated by exposure to wind, &c. Usually there is swelling at the site of the sac. Often the caruncle and neighbouring parts of the conjunctiva are inflamed. On pressure over the sac, fluid regurgitates through the puncta, or more rarely passes down into the nose. The fluid may be tears, mucus, or muco-pus; the swelling is often called a *mucocele*. Bacteriological examination of the fluid demonstrates the presence of an extraordinary number of bacteria—staphylococci, pneumococci, streptococci, &c. Of these the pneumococcus is very frequently present in virulent form. This fact is of supreme importance, since it explains the frequency with which hypopyon ulcer arises in these cases, and the danger of panophthalmitis if any intra-ocular operation is undertaken. Dacryocystitis is a constant menace to the eye, since minute abrasions of the cornea are of almost daily occurrence, and such an abrasion is liable at any moment to become infected and give rise to an hypopyon ulcer.

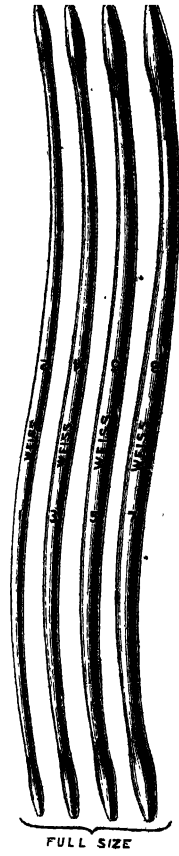


FIG. 353.—Couper's lacrymal probes.

Chronic dacryocystitis is commonly attributed to the effects of stricture of the nasal duct. It is by no means certain that the stricture is primary in all these cases; it is not unlikely that it sometimes results from the inflammation of the sac or from the treatment applied to remedy the dacryocystitis. It would appear, however, that in the majority of cases the primary infection is nasal in origin and there are many undoubted cases in which intractable dacryocystitis has been cured by treatment of a coincident nasal inflammation. Obstruction to the lower end of the nasal duct may be caused by

the pressure of nasal polypi, an hypertrophied inferior turbinate bone, extreme deviation of the septum, and so on.

Untreated chronic dacryocystitis never undergoes spontaneous resolution. The condition tends to progress, the walls of the sac ultimately become atonic, the contents never being evacuated except by external pressure. In any case an acute inflammation may arise, a lacrymal abscess being formed. This sequel may be caused by treatment, an abrasion of the epithelial lining leading to infection of the pericystic tissues.

FULL SIZE

Fig. 354. — Lüer's syringe, with nozzle, for syringing the lacrymal passages.

The patients are usually elderly, and such as are exposed to dirt in the course of their daily occupations. Want of personal cleanliness is probably an important factor. Dacryocystitis may, however, occur in the new-born. In these cases it is generally due to adhesion of the epithelial lining, or to imperfect canalisation of the epithelial cord in which the nasal duct is formed. The careful passage of a small probe *once* will cure these cases. Occasionally dacryocystitis in babies is extremely intractable: I am of the opinion that most of these cases are tuberculous or syphilitic, usually originating in caries of the surrounding bones.

Tubercle of the lacrymal sac also occurs in adults as a rare form of dacryocystitis.

*Treatment.* In the new-born penicillin drops should be ordered, and minute directions should be given for expressing the contents of the sac, which should be done very frequently. Many cases will be cured by this treatment. If it fails after a fortnight, an anæsthetic should be given and a small probe passed down the nasal duct, the greatest care being exercised to avoid injuring the walls of the duct. It is unnecessary to slit up the canaliculus. The punctum and canaliculus are dilated with a Nettleship's dilator. A small probe (No. 1 or 2) is inserted vertically downwards into the canaliculus, then passed gently but firmly inwards until the point is felt against the lacrymal

bone. The probe is then rotated upwards and towards the middle line, and pushed down the nasal duct until it touches the floor of the nose. It should be remembered that the duct is short in the new-born. The force required is quite slight if rightly applied in the line of the duct (*vide* p. 649). Since much harm may be done by bad probing, these cases should be treated by an expert.

In adults the conjunctival sac should be anæsthetized. The punctum is dilated and the sac syringed out with a lacrymal syringe (Fig. 354). A moderately fine straight cannula should be used. The point is inserted into the canaliculus; it need not pass into the sac. Two or three syringefuls of penicillin solution (1,000 units per c.c.) are passed. Probably the whole of the fluid will regurgitate through the upper canaliculus. The operation should be repeated every day for a fortnight or

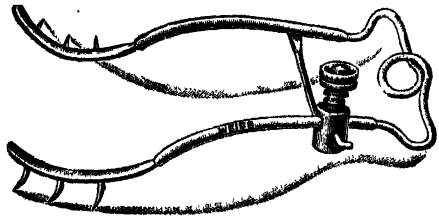


FIG. 355.—Briggs's retractor.

longer. In the majority of cases the fluid will pass freely down into the nose in a few days. When this occurs the syringing should be repeated at constantly increasing intervals. A great number of previously untreated cases can be cured in this manner. The patient should be told to squeeze out the contents of the sac frequently in the intervals between syringing.

The rationale of this treatment depends upon the fact that the walls of the sac and upper part of the duct are inflamed. The swollen mucous membrane prevents the fluid in the sac from passing into the nose. The treatment reduces the swelling and restores the communication.

In the meantime the condition of the nasal fossæ should be thoroughly investigated by an expert, and any pathological condition likely to cause inflammation or obstruction of the nasal duct treated.

If no cause is discovered in the nose either excision of the

lacrymal sac or the establishment of permanent drainage into the nose by dacryocystorhinostomy must be undertaken. The radical operation, properly performed, completely removes the disease, with a minimum of inconvenience to the patient.

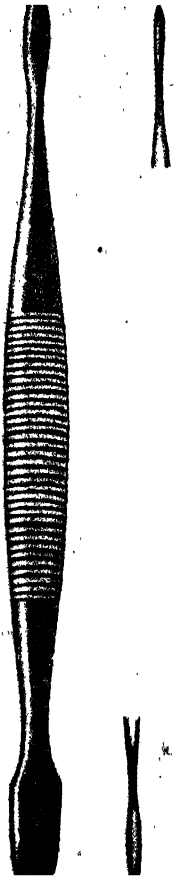


FIG. 356.—Stallard's lacrymal dissector.

Many surgeons treat chronic dacryocystitis by probing. The canaliculus is slit up, and probes of increasing calibre are passed down the nasal duct into the nose (Fig. 353). The objections to this method of treatment are—(1) it is impossible to probe the swollen and inflamed duct without injuring the walls; (2) such injury may lead to infection of the surrounding tissues and an acute cellulitis; (3) in any case healing of the abrasions is accompanied by the formation of connective tissue, which contracts when it organises and leads to fibrous stricture instead of obstruction by swollen mucous membrane; (4) probing is always painful, and when once begun has to be continued for a prolonged period; (5) most cases are alleviated only temporarily, fresh courses of probing being required at intervals. Protargol should never be injected immediately after slitting up the canaliculus or probing. If an abrasion of the mucous membrane has been caused the protargol may be injected into the subcutaneous tissues and violent cellulitis follows. Orbital cellulitis and atrophy of the optic nerve have been known to result from neglect of this rule. Even if these serious results do not follow there is unsightly and permanent staining of the skin. These facts are proof of the injury done to the mucous membrane by probing.

*Excision of the Lacrymal Sac* (*Dacryocystectomy*) is performed as follows. Instruments required: knife, fixation forceps, Brigg's retractor (Fig. 355), blunt dissector (Fig. 356) blunt-pointed conjunctival scissors, curette, 4 mosquito pressure forceps, 1 fine catgut ligature and 1 suture,

1 gossamer horse-hair suture, white silk suture, needle holder, 2 small claw retractors, canaliculus rasp, punctum dilator, Couper's lacrymal probes, and lacrymal syringe.

The operation can be performed with local anæsthesia. Four drops of pantocain (1 per cent.) are instilled and novocain or novutox (2 per cent.) with adrenaline is injected through the skin just above the fundus of the sac and along the upper canaliculus. A second injection is made along the lacrymal crest over the sac, and is carried deeply along the floor of the orbit where the sac joins the nasal duct. It is also necessary to

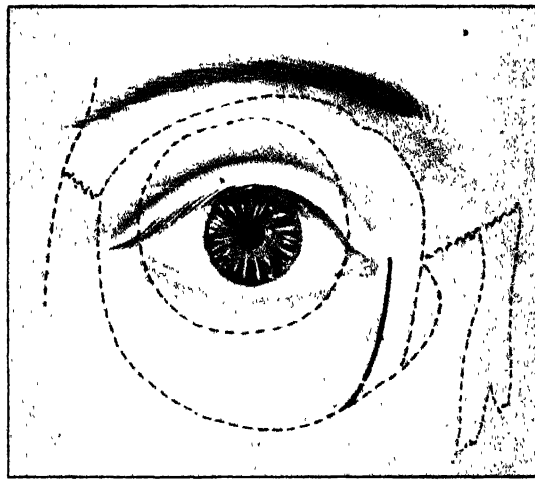


FIG. 357.—Incision for excision of the lacrymal sac. The broken lines indicate the bones and orbital margin; also the limits of the conjunctival sac.

inject 3 minims of novutox into the skin of each lid 3 mm. from the centre of the lid margin. The ipsilateral nasal fossa is sprayed with cocaine and adrenaline, and may be packed with ribbon gauze soaked in an oily solution of the same drugs.

The canaliculi are fully dilated, and the lacrymal sac irrigated with warm saline. Five minims of sterile melted wax impregnated with methylene blue may be injected so as to assist in the identification of the sac. The lids are temporarily closed with mattress sutures passed through the skin 3 mm. from the centre of the lid margins in order to avoid the danger of an infected abrasion.

An assistant stretches the skin by moderate traction with one finger at the outer canthus and the other on the bridge of the nose. A curved incision is made, beginning 2 mm. above the medial palpebral ligament and 3 mm. to the nasal side of the inner canthus; it is carried vertically downwards for 4 mm., and then outwards along the line of the anterior lacrymal crest to a spot 2 mm. below the inferior orbital margin. The skin of the temporal edge of the incision is

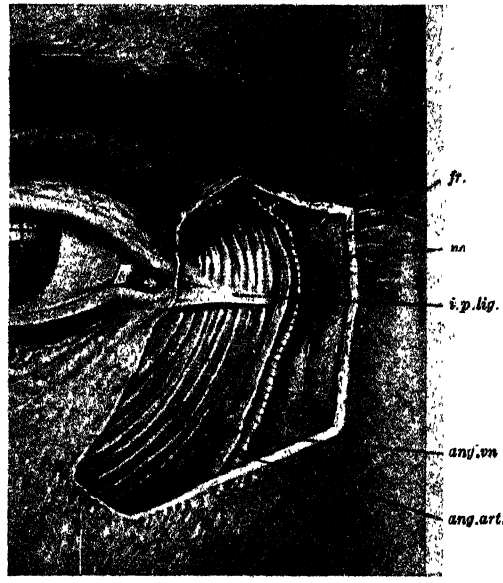



FIG. 358.—Relations of angular artery and vein. *ang.art.*, angular artery; *ang.vn.*, angular vein; *i.p.lig.*, internal palpebral ligament; *nasal*, nasal bone; *fr.*, frontal process of superior maxilla. (Eugene Wolff. "Anatomy of the Eye and Orbit." Lewis, London.)

undermined for 2 or 3 mm., but not that of the nasal edge, owing to risk of wounding the angular vein or its branches. The orbicularis is split in the line of the incision, and Briggs's retractor inserted so as to retract it with the skin. The lacrymal fascia is exposed and incised along the anterior lacrymal crest, thus bringing the bluish sac into view. This is freed from the bone on the nasal side by the blunt dissector (Fig. 356) and from the palpebral ligament, &c., by careful dissection until it remains attached only below to the nasal duct. The sac is

drawn forwards and twisted two or three times in pressure forceps until it tears away from the duct. The upper end of the duct is curetted, and a Couper's probe passed down into the nose. The lids are now released and the epithelium lining the canaliculi removed by a canaliculus rasp. The orbicularis is sutured with catgut, and the skin incision by a continuous subcuticular suture. The eye is irrigated and a drop of penicillin (1,000 units per c.c.) instilled. A pyramid-

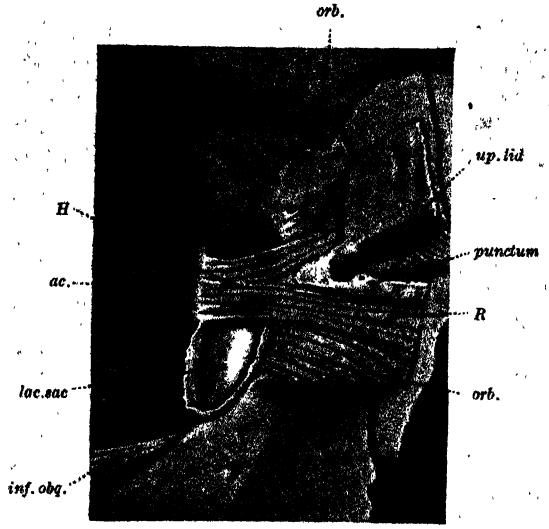



FIG. 359.—Lacrymal sac. *H.*, Horner's muscle; *lac.*, lacrymal bone; *lac. sac.*, lacrymal sac; *inf.obq.*, inferior oblique; *orb.*, orbicularis. (Eugene Wolff. "Anatomy of the Eye and Orbit." Lewis, London.)

shaped gauze dressing, with its apex against the wound is applied firmly.

*Dacryocystorhinostomy* is a more difficult operation, and is only suitable for cases of young or middle-aged adults with dacryocystitis of comparatively recent origin. The early steps of the operation are the same as for excision of the sac. An opening is then made through the lacrymal bone into the nose. The nasal mucosa is freed and the lacrymal sac incised, so as to fashion two panels. Fig. 361 shows the suturing of the posterior panels. The anterior panels are similarly sutured, thus covering the bony aperture with mucous membrane. When successful this operation restores a quasi-normal excretion of the tears into the nose, but

unless the opening is large and local infection has been controlled, the opening from the sac through the bone into the nose may become blocked with granulation tissue.

There is no objection to removing both lacrymal sacs at the same operation if there is bilateral mucocele. When the operation is satisfactorily performed there is no regurgitation on pressure over the scar. If after a week or two there is still some regurgitation, part of the mucous membrane has been left behind, and the operation must be repeated. Usually it is the fundus of the sac which has been left. This lies above

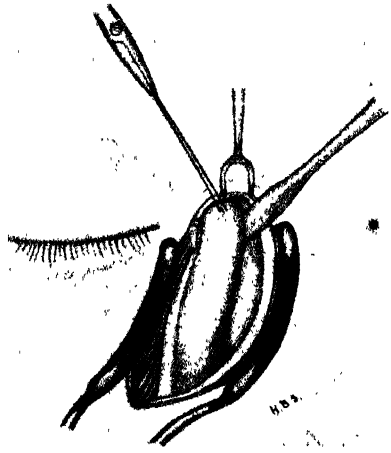


Fig. 360.

the palpebral ligament; hence the advisability of dividing the ligament in some cases in order that a good view may be obtained. Sometimes regurgitation is due to leaving the mucous membrane of the upper part of the nasal duct; it will not occur if the duct is well curetted.

In all cases of cataract in which there is a mucocele the lacrymal sac should be excised as a preliminary to extraction. Only some weeks after this operation, when there is no trace of regurgitation, is it permissible to proceed with the extraction. A more difficult problem is the presence of a mucocele in a case of acute glaucoma. Here immediate iridectomy may be indicated and admits of no delay. In these cases the sac must

be completely isolated from the conjunctival sac. This is best effected by passing a ligature round each canaliculus and tying it firmly. Some surgeons cauterise the punctum, thus sealing it up with a cicatrix. Either procedure may be followed by the development of a lacrymal abscess, but in the meantime the iridectomy wound has probably healed, and in any case the pus is evacuated through the skin and not into the conjunctival sac.

Epiphora usually persists for a time after excision of the sac. This is due to the chronic conjunctivitis set up by the mucocele: since, as already stated (p. 649), under normal conditions the tears evaporate from the surface of the globe. Hence the post-operative epiphora should be treated by

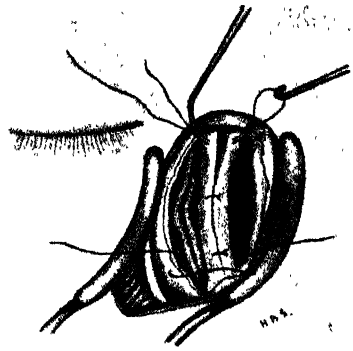


FIG. 361.

astringent lotions, &c.; in no case is it necessary in my experience to remove the lacrymal gland, as has been advocated. Epiphora will, however, still occur on exposure to wind, &c., and this cannot be avoided.

**Lacrymal Abscess** may be due to acute dacryocystitis or to suppuration starting in the pericystic tissues. The skin over the sac becomes red and swollen. The redness and swelling rapidly extend to the lower lid and upper part of the cheek, so that the condition may be easily mistaken for erysipelas. There is severe pain, and often some fever. The abscess usually points below and to the outer side of the sac owing to gravitation of the pus to the margin of the orbit. If it opens spontaneously pus continues to be discharged for some time, and a permanent fistula is likely to result.

*Treatment.* General treatment by bacteriostatic drugs penicillin or sulphonamides) should be instituted at once (*vide* p. 695). If seen at the beginning of the process an attempt may be made to draw the contents of the sac into the nose by cocaining the ipsilateral nasal fossa and inserting a tampon soaked in adrenaline (1 in 2,000) over the opening of the nasal-lacrymal duct. An injection of 5 minims of adrenaline (1 in 2,000) is made into the lacrymal sac. In some early cases the muco-pus can then be coaxed down the nasal duct.

Hot bathing should be persevered with and incision should not be resorted to unless the abscess is pointing under the skin, in which case it should be opened by a small incision, the pus gently squeezed out, a piece of rubber-glove drain inserted, and a dressing of penicillin and sulphonamide powder applied.

If the discharge continues for a long period the cavity should be well curetted and again drained. Sometimes the epithelial lining of the sac is destroyed by the purulent inflammation, the sac is permanently destroyed, and the cure is complete. In other cases some of the mucous membrane escapes destruction, and a fistula may follow. It may sometimes be closed by cauterising the edges with the galvanocautery, but it is better to re-open the sac along a director introduced through the fistula and extirpate the remnants. This procedure should not be adopted until several weeks after the acute inflammation has subsided.

**Stricture of the Nasal Duct** has already been referred to incidentally. It is probable that most intractable fibrous strictures are caused by probing, though it cannot be asseverated that they may not arise spontaneously as the result of destruction of the epithelium by extension of inflammation from the nose or lacrymal sac. Occasionally bony strictures occur, usually caused by fractured maxilla, inflammation of the antrum, or caries.

*Treatment.* The usual treatment of stricture of the nasal duct is dilatation with probes. The objections to this treatment have been mentioned. I advocate excision of the lacrymal sac in these cases.

# CHAPTER XXXIII

## Diseases of the Orbit

It is unnecessary to describe the anatomy of the orbit and its contents here. The student is recommended to revise his knowledge of the subject, paying special attention to the relations of the nasal cavities and their accessory sinuses, and to the communications with the interior of the cranial cavity by way of the optic foramen and sphenoidal fissure. The intimate adhesion of the dural sheath of the optic nerve to the walls of the optic foramen is of great pathological importance, and the relations of the intraorbital to the intracranial circulation must be thoroughly appreciated. The eye is slung in position in the orbit by fascia, one sheet of which, Tenon's capsule, forms a socket in which the globe moves. This, with the sclerotic, forms a lymphatic space, lined completely with endothelium. The extrinsic muscles of the eye do not perforate this capsule, but invaginate it, the fascia being reflected from their surfaces.

The normal position of the eye is such that a straight-edge applied vertically to the middle of the upper and lower margins of the orbit just touches the closed lids over the apex of the cornea. There are individual variations which are of no pathological importance when symmetrical; in all cases of doubt the two sides should be compared.

Abnormal protrusion of the globe is called *exophthalmos* (more accurately, *exophthalmia*) or *proptosis*. It is much commoner than abnormal retraction or *enophthalmos*. The former condition is due to many causes, among which increase in the orbital contents and loss of tone of the extrinsic ocular muscles are the most important. Slight prominence of the eyes accompanies high myopia, paralysis of the extrinsic muscles, stimulation of Müller's muscle by cocaine, and as an idiosyncrasy, especially in very obese people. Unilateral exophthalmos occurs in orbital cellulitis from any cause, thrombosis of the orbital veins with or without implication of the cavernous sinus, arterio-venous aneurysm, tumours of the orbit and its contents, and orbital hæmorrhage or

emphysema. Bilateral proptosis occurs in exophthalmic goitre, the later stages of thrombosis of the cavernous sinus, empyema of the accessory sinuses of the nose, symmetrical orbital tumours (lymphoma, pseudoleukæmia), and as a result of diminished orbital volume in oxycephaly or "tower-skull" and leontiasis ossium. Enophthalmos is generally due to severe injury in which the orbital bones are fractured, or to orbital cellulitis with mechanical retraction by fibrous tissue. Slight degrees of exophthalmos or enophthalmos are best diagnosed by the test with a straight-edge. Accurate estimates of the amount can be obtained only by special mechanical devices (exophthalmometers). A convenient test is the following: The patient is seated, the surgeon standing behind him. The surgeon holds the patient's head in such a manner that he looks straight down the nose. He then rotates the head backwards until he can just see the apex of one cornea. If he can see more of the other cornea, that eye is relatively proptosed.

### Orbital Inflammation

**Periostitis** is not uncommon, particularly affecting the margin. It is most often due to injuries, extension of inflammation from neighbouring parts, tubercle or syphilis. Tuberculous periostitis is most frequent in children, syphilitic in adults: in the former, caries of the bone results; the latter is gummatous. In traumatic cases the margin is naturally most affected, but a traumatic element is often an exciting cause in the other cases, so that in them also the margin most frequently suffers.

When situated at the margin, the inflamed part is swollen and tender; the swelling is intimately connected with the bone, so that it cannot be moved over it. Syphilitic cases usually respond well to treatment. The other types generally go on to suppuration. An abscess is formed, and when it discharges or is opened rough bone can be felt with a probe. In tuberculous cases particularly a fistula may result, the edges of the aperture being bound down to the bone, so that a depressed cicatrix is formed. The fistula remains open until all the necrosed bone is extruded. The cicatrisation may lead to displacement of the lid—ectropion, lagophthalmia, and so on.

Periostitis of the deeper parts of the orbit causes less definite signs. There is more pain of a deep-seated character. There may be proptosis with deviation in the direction of the

eye. In the case of gumma the roof of the orbit is generally involved, the deviation of the eye is downwards, and there is rapid loss of movement owing to involvement of the extrinsic muscles. There is severe supraorbital neuralgia, which is worse at night. Often the true nature of the disease is only discovered by an exploratory operation, or by the evacuation of pus. The case may present all the features of orbital cellulitis (*q.v.*). If the roof of the orbit is involved, the pus may discharge into the cranial cavity, life being endangered by meningitis or cerebral abscess.

*Treatment* is determined by the ætiological factor. In most cases general treatment by penicillin or, if indicated, sulphonamides has a dramatic effect. In syphilitic cases, mercury and iodide of potassium are pushed rapidly. In traumatic cases, if suppuration supervenes, the abscess is opened, hot fomentations being applied previously if necessary. In tuberculous cases an incision should be made early and any carious bone removed, care being taken not to encroach upon the cranial cavity.

In deep-seated periostitis an exploratory operation may be necessary, and should not be too long delayed. An incision is made through the skin at the margin of the orbit, the knife being passed cautiously deep into the orbit along the wall. The site of the incision is determined by the signs present. Sinus forceps are passed down the track of the wound and opened. The greatest care should be exercised to avoid unnecessary damage to the orbital contents, and this is best accomplished by keeping closely to the bony walls. Special care must be taken not to injure the pulley of the superior oblique. If pus is found, a small drainage tube or a strip of rubber glove or cyanide gauze is inserted. In periostitis of the inner wall, the bone may be extensively diseased. Severe operations, involving the opening of the frontal or ethmoid sinuses, may be essential, with or without drainage through the nose. These cases often do remarkably well. They are usually tuberculous, and occur most frequently in children. Exploration of the orbit in children is much more difficult than in adults. The eye is relatively much larger in comparison with the size of the orbit, so that there is very little room between the globe and the orbital wall. In rare cases it may be advisable to perform Krönlein's operation (*vide* p. 675).

**Orbital Cellulitis** is purulent inflammation of the cellular tissue of the orbit. It is due to deep injuries, especially those

with retained foreign body, or septic operations, *e.g.*, enucleation of the eyeball; extension of inflammation from neighbouring parts, especially the nasal sinuses and teeth; facial erysipelas; metastasis in pyæmia, meningitis, infective fevers, &c.

There is great swelling of the lids, with chemosis. The eye is proptosed, and its mobility impaired. Pain is severe, increased by movement of the eye or pressure upon it. Fever is present, and cerebral symptoms may arise. Movement of the eye is painful, and there may be diplopia owing to limitation of movement. Vision may not be affected, or it may be reduced owing to retrobulbar neuritis. The fundus is difficult to examine; it may be normal or show engorgement of the veins and optic neuritis, passing later into optic atrophy. An abscess is formed which usually points somewhere in the skin of the lids near the orbital margin, or it may empty into the fornix conjunctivæ. Panophthalmitis may supervene. There is grave danger of extension to the meninges and brain, leading to a fatal issue from purulent meningitis or cerebral abscess. Thrombosis of the cavernous sinus (*q.v.*) may result from orbital cellulitis, and is always difficult to diagnose from it.

*Treatment.* General treatment by the bacteriostatic drugs should be instituted at once (penicillin or sulphonamides (*vide* p. 695)), and if instituted reasonably early, resolution may rapidly follow. Hot bathings, and medical diathermy, if available, may be applied, but must not be relied upon too long. An early incision as in orbital periostitis (*q.v.*) is imperative. Even if pus is not reached, the tension is relieved and a track is prepared for its evacuation. If the source of infection is obscure, the nose and other likely seats must be investigated, and the primary focus treated. The administration of sulphonamides by the mouth may be helpful.

**Thrombosis of the Cavernous Sinus** may be due to extension of thrombosis from various sources.

The anatomy of the venous channels which communicate with the cavernous sinus is of prime importance for the comprehension of thrombosis affecting it (Figs. 362, 363). The superior and inferior ophthalmic veins enter it in front and the superior and inferior petrosal sinuses leave it behind. It communicates directly with the pterygoid plexus through the middle meningeal veins and the vein of Vesalius, and indirectly through a communicating vein from the inferior ophthalmic to the pterygoid plexus. The anastomoses of the ophthalmic veins with the frontal and angular

open up a communication with the face. Labyrinthine veins opening into the inferior petrosal sinus afford a communication with the middle ear. Numerous tributaries throw it into direct or indirect communication with most parts of the cerebrum. The mastoid emissary vein places the sinus in communication with the subcutaneous tissues behind the ear through the lateral sinus and superior petrosal sinus; it is this communication which is of great diagnostic importance, since swelling behind the ear may decide the question of thrombosis in each direction along them. The sinus of one side communicates with that of the other by two (or sometimes three) transverse sinuses which surround the pituitary body.

Infection may occur *vid* the orbital veins—*e.g.*, erysipelas and septic wounds of the face, orbital cellulitis, and mouth and

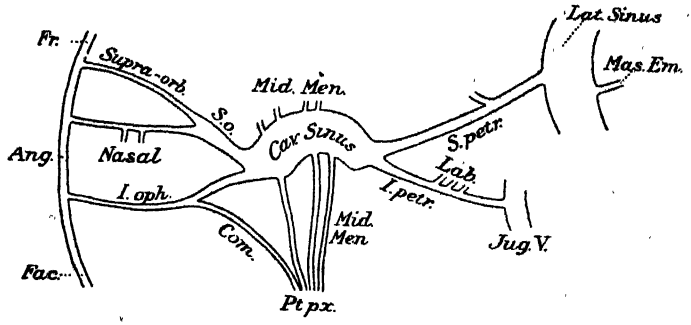



FIG. 362.—Tributaries of the cavernous sinus (lateral view).

pharynx; from the ear, nose and accessory sinuses; or as a metastasis in infectious diseases or septic conditions.

The patient presents almost the same symptoms and signs as in orbital cellulitis. If in addition there is œdema in the mastoid region behind the ear the diagnosis is certain, for this is due to thrombosis of the emissary vein. A further point of diagnostic importance is transference of the symptoms to the opposite eye, which occurs in 50 per cent. of cases, whereas bilateral orbital cellulitis is very rare. The first sign is paralysis of the opposite external rectus, and this sign should be carefully watched for in any suspicious case of inflammatory unilateral exophthalmos. It must be remembered, however, that thrombosis of the sinus may be a complication of cellulitis.

There is severe supraorbital pain, owing to implication of

the branches of the ophthalmic division of the fifth nerve, and the motor ocular nerves are paresed or paralysed. In the later stages the eye is immobile, the pupil dilated, and the cornea anæsthetic. Proptosis occurs in nearly all cases, but is of late onset in those of otitic origin.

It is commonly stated that the retinal veins are greatly engorged, but in many cases this is certainly not true. When it occurs it is usually accompanied by pronounced papillitis, and both signs indicate extensive implication of the orbital veins and tissues. Simultaneous thrombosis of both cavernous sinuses, with proptosis and papillitis, occurs in diseases of

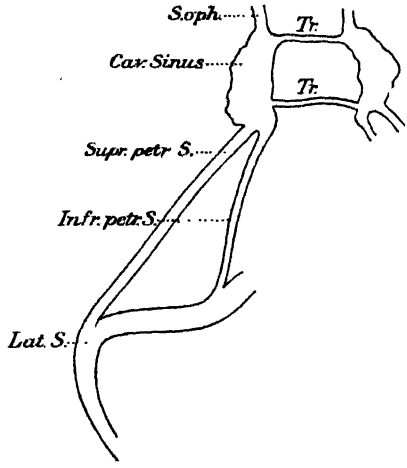



FIG. 363.—Tributaries of the cavernous sinus (from above)

the sphenoidal sinuses. Typical papilloedema is commonest in otitic cases, and indicates meningitis or cerebral abscess: it is bilateral, and more pronounced on the side of the aural lesion.

Thrombosis of the cavernous sinus is accompanied by rigors, vomiting, and severe cerebral symptoms. Before the advent of the modern bacteriostatic drugs the patient usually died, but early penicillin therapy may bring about resolution.

**Tenonitis** is inflammation of Tenon's capsule: it may be serous or purulent. There is exophthalmos straight forwards, with limitation of movement of the globe and pain on attempted movement. There may be some œdema of the lids, and chemosis. It may occur in severe iridocyclitis, and is constant in panophthalmitis. It may also follow tenotomy,

&c. Simple serous tenonitis is rare, and has been attributed to influenza, gout, rheumatism, &c.

*Treatment* consists in general treatment by the bacteriostatic drugs (penicillin or sulphonamides) together with the application of hot bathings and the evacuation of pus, if it forms. When it occurs as part of panophthalmitis, &c., it requires no special treatment.

### Distension of the Accessory Sinuses of the Nose

The accessory sinuses of the nose—the frontal, ethmoidal and sphenoidal sinuses, and the antrum of the superior maxilla

Fig. 364.—Distension of the frontal sinus.

—are separated from the orbit only by thin plates of bone. The orifices which form the communication between these cavities and the nose are liable to become occluded by catarrh, polypi, neoplasms, &c. The normal sero-mucous discharge is thus unable to drain into the nose. The cavities become distended with fluid, and owing to the presence of pyogenic organisms pus may be formed. The treatment of the conditions thus set up cannot be considered part of the functions of the ophthalmic surgeon, but he must be prepared to diagnose them since they not infrequently appear for the first time in the ophthalmic clinic. This is particularly the case in distension of the frontal, ethmoidal and sphenoidal sinuses. Of these the frontal sinus suffers most often.

Distension or empyema of the frontal sinus causes bulging at

the upper and inner part of the orbit (Fig. 364). There may be some proptosis and displacement of the eyeball downwards and outwards, but these features are more marked when the ethmoidal sinus is involved. Œdema of the upper lid or slight ptosis may be the only external sign. There is considerable pain and tenderness, with severe headache. There is often discharge from the nostril of the same side, or manifest disease of the nasal cavities. Owing to erosion of the walls of the sinus the fluid may extend under the periosteum, causing bulging into the posterior part of the orbit. It may escape into the opposite sinus and through the infundibulum of that side; or it may rupture into the orbit, through the skin, forming a sinus, or even into the cranial cavity. Orbital cellulitis may be set up in this manner.

The frontal sinus is not developed until about the sixth year; the disease occurs most commonly between twenty-five and thirty, and more cases occur in men than in women.

Treatment of distended frontal sinus consists in providing free discharge of the contents through the nose. In most cases a radical cure is effected only by laying open the sinus, scraping away completely the diseased mucous membrane, and passing a drain down into the nose. The disease is tedious to treat and much disfigurement may follow. Displacement of the pulley of the superior oblique may lead to diplopia, which may persist for several months or permanently.

Distension of the ethmoidal cells by polypi, new growths or inflammatory products may also cause bulging into the orbit and displacement of the globe. Diplopia, chemosis, venous engorgement and ptosis may be caused. Ethmoiditis is usually associated with nasal discharge. It may give rise to orbital cellulitis, or in less severe cases to retrobulbar neuritis. The latter is probably more commonly associated with inflammation and distension of the sphenoidal cells, which lie in close proximity to the optic nerve, being sometimes separated from it by a very thin lamina of bone. In doubtful cases help may be afforded by a skiagram. It has already been mentioned that the accessory sinuses of the nose are not infrequently the foci from which toxins are disseminated, leading to iridocyclitis and other metastatic septic processes in the eye (*vide* pp. 274, 343).

### Injuries of the Orbit

Injuries to the soft parts usually arise from penetration of a foreign body, which may be retained. The lids and eyeball

are frequently implicated. The signs depend upon the particular structures injured. In most cases there is considerable hæmorrhage; as the blood does not find a ready exit exophthalmos may result. Extravasation of blood under the conjunctiva and into the lids is common. Hæmorrhage may result from pressure with forceps at birth. It also occurs in some cases of fracture of the base of the skull. Paralysis of extrinsic muscles may be due to direct injury or to injury of the motor nerves. The optic nerve may be severed or retrobulbar neuritis may ensue; in either case atrophy, involving the optic disc, follows (*vide* p. 404), or atrophy may follow hæmorrhage into the sheath of the nerve. The nerve may be divided either posterior to, or rarely, anterior to the entrance of the central retinal vessels. Avulsion of the disc, with the formation of a traumatic "coloboma" or "conus" of the disc, may occur, even without rupture of the sheath of the nerve. The eyeball may be perforated or contused (*vide* p. 437) or dislocated *en masse*. Dislocation forwards between the lids occurs most often when the blow is directed from the outer side, where the orbital margin affords least protection. Insane patients sometimes enucleate their eyes by gouging them out with their fingers. Sight is not necessarily lost after dislocation forwards. Retained foreign bodies are extremely liable to set up suppuration and orbital cellulitis (*q.v.*).

Injuries to the bone most commonly affect the margin of the orbit. Fractures in this locality are easy to diagnose from the unevenness of the margin, sensitiveness to pressure, and sometimes crepitation. Emphysema (*q.v.*) may occur. The soft parts may be injured by splinters of fractured bones. Deep fractures may be caused by penetrating wounds or by severe contusions, falls, &c. Fracture of the base of the skull may involve one or both optic foramina, in which case the optic nerve is often injured, or pulsating exophthalmos (*q.v.*) may ensue. Blindness without ophthalmoscopic signs may be caused in this manner: atrophy of the disc follows in three to six weeks (*vide* p. 404).

Gunshot wounds of the orbit, without direct involvement of the eye, frequently produce concussion changes which appear ophthalmoscopically as coarse tracks of white exudate in the retina and choroid, large blot-like hæmorrhages, and multiple small choroidal tears. These resolve into dense white scarred areas fringed with pigment, with finer pigmentary disturbance elsewhere in the fundus. The site may give an indication of the direction of the track of the missile and assist in localising a

retained intracranial foreign body. Both eyes should be examined, as the missile may have traversed both orbits.

*Treatment.* If there is a wound it must be cleansed, and, if necessary, probed: it should be dusted with penicillin-sulphonamide powder and a prophylactic course of general penicillin treatment given if indicated. Absorption of extravasated blood is often very slow. The treatment of a retained foreign body depends upon its situation and the probability of suppuration occurring. If the foreign body cannot be extracted with ease a skiagram should be taken. If the position is such that very serious manipulation would be requisite for removal, and if there is evidence that the substance is aseptic, expectant treatment may be adopted. If suppuration occurs the foreign body must be removed and the case treated as one of orbital cellulitis (*q.v.*).

### Tumours of the Orbit

Orbital tumours are rare. Benign growths include dermoid cyst, dermo-lipoma (*vide* p. 195), angioma, osteoma (Fig. 365), plexiform neuroma (*vide* p. 647), meningo-encephalocele. Of these, dermoid cysts appear as swellings under the lid, usually at the upper and outer angle; they contain sebaceous material derived from sebaceous glands in the walls, which are lined with epithelium and possess hair follicles; they sometimes contain fœtal remnants (teratoid cysts). Clinically they may be mistaken for meningo-encephaloceles, which usually occur at the upper and inner angle, where there are most sutures between bones. In the latter—(1) the tumour is immovably attached to the bones; (2) the hole in the bone may be palpable; (3) pulsation, synchronous with respiration and the pulse and increasing on straining,

Fig. 365.—Orbital osteoma. (Tweedy.)

can be seen; (4) pressure may cause diminution in size due to fluid being pressed back into the cranium; (5) exploratory puncture produces clear fluid with the characteristics of cerebro-spinal fluid. Osteomata start usually from the frontal bone (Fig. 365); they are intensely hard and often large, producing great displacement of the globe.

Malignant tumours of the orbit are usually sarcomata, though carcinoma derived from the lacrymal gland (*vide* p. 650) or by extension from the nasal mucous membrane also occurs.

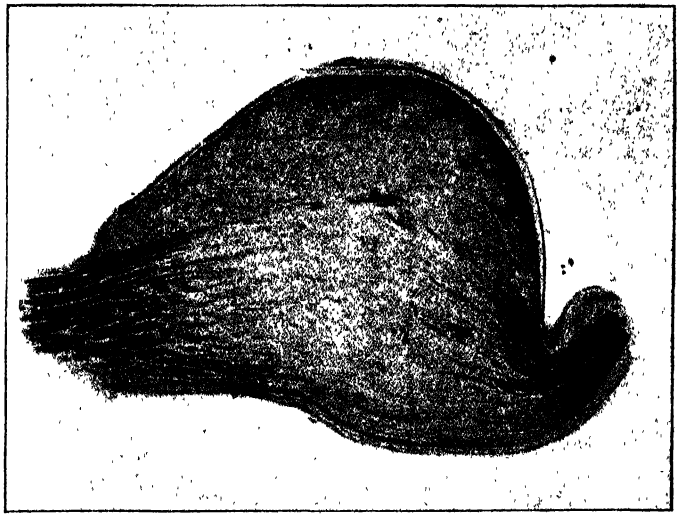


FIG. 366.—Intradural tumour of the optic nerve. (Mayou.) Longitudinal section, section stained by Weigert-Pal, to show distribution of the nerve fibres.

All types of sarcoma, including endothelioma and myeloid sarcoma, may occur. The small round-celled growths include cases of lymphoma, leukæmic tumours, chloroma, &c.

Primary tumours of the optic nerve manifest themselves clinically as orbital tumours. They are rare, and are found on anatomical examination to consist of two groups—neuromata and meningiomata. Of these the former are more numerous (Fig. 366). Selective staining shows that they are nearly all true gliomata springing from the neuroglia; a few are endotheliomata or fibromata, in the proportions roughly of 20 : 4 : 1. They spread slowly within the sheaths, and death

is due to intracerebral extension. Meningiomata are usually endothelial, springing from the arachnoid and endothelial lining of the dura. They infiltrate the nerve and may spread to the orbit. Rare extradural growths springing from the dura are chiefly fibrous in structure (fibromata); and may fill the orbit (Fig. 367). All these optic nerve tumours are most common in children and are locally malignant, but show little or no tendency to metastasis.

Most orbital tumours cause proptosis, which is very rarely straight forwards except in the case of optic nerve tumours. This is an important diagnostic feature. The exophthalmos increases slowly and gradually, and is nearly always unilateral: in rare cases of lymphoma it is bilateral. The mobility of the eyeball is impaired in the direction towards the position of the tumour. There is usually diplopia from this cause. Papillœdema may be present, especially with optic nerve tumours. Optic atrophy from pressure on the nerve is common in the other forms. The tumour may be palpable by the finger pushed back between the globe and the orbital wall. The lymphatic glands are seldom affected.

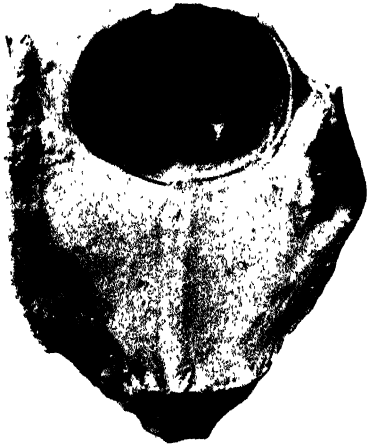


FIG. 367.—Extradural tumour of the optic nerve (× 1·4).

Careful examination of neighbouring parts—nose, antrum, mouth (especially the naso-pharynx, and line of the teeth)—must be made to determine whether the invasion of the orbit is secondary or whether the growth is primarily orbital. In doubtful cases an X-ray examination should be made.

*Treatment.* An exploratory operation and removal of a portion of the growth for microscopic examination may be a necessary preliminary to radical treatment. It may be feasible to remove dermoid cysts and some other benign tumours without injury to the globe, though its mobility is likely to be impaired in extensive operations. As already mentioned, many malignant orbital growths show little

tendency to metastasis, so that their treatment may be more conservative than is usual in other parts of the body. Thus it is possible in some cases of optic nerve tumour to remove the growth while retaining the eyeball. This can be effected by *Krönlein's operation*, which is also of utility as an exploratory procedure in some cases. In it a semilunar incision is made vertically just outside the outer canthus, the convexity being directed forwards. The bone is chiselled through at the upper and lower outer angles of the orbit, and bone, muscle and skin are reflected backwards in one flap. The posterior part of the orbit is thus exposed in a manner which is impossible by any other method. The greatest care must be exercised that infection does not occur, since the spongy bone is laid open, and there is also danger of meningitis. An alternative method of approach which gives wide access to the apex of the orbit is by the intra-cranial transfrontal route.

In the case of more malignant types of tumour their complete removal is imperative at all costs, and the eye, which may be quite normal, may have to be sacrificed. In these cases, as well as in recurrence or in orbital extension of malignant intraocular growths (retinoblastoma, sarcoma of the uveal tract), it may be necessary to remove the whole contents of the orbit.

In *exenteration of the orbit* the lids may be retained if they are not implicated in the growth, but the free margins, carrying the cilia, should always be removed. If it is not done the lashes are troublesome when the lids become retracted into the orbit, as invariably follows. If the lids are removed the incision is carried through the skin at the margin of the orbit in its whole circumference. The orbital contents are separated from the walls by a periosteal elevator, so that they remain attached only at the apex of the orbit. The pedicle is then severed with strong scissors, or preferably by diathermy, thus avoiding hæmorrhage. At a later stage it may be advisable to apply Thiersch grafts to the walls, since the lids and conjunctiva never afford sufficient epithelial covering, and the extension of the epithelium over so large a surface is a tedious process.

Some of these tumours respond to irradiation and radium treatment by shrinking, but the ultimate results are usually disappointing. Recurrence in the orbit should, however, be treated by these means.

### Special Forms of Exophthalmos

**Exophthalmic Goitre** (*Syns.—Graves's or Basedow's Disease*) is one of the commonest causes of exophthalmos (Fig. 368). The symptom complex includes, besides proptosis, enlargement of the thyroid gland, tachycardia, muscular tremors, and raised basal metabolism. The proptosis is almost always bilateral and may be extreme, leading to lagophthalmia and its deleterious consequences (*vide* p. 225). There is a peculiar stare, with retraction of the upper eyelid, so that there is an unnatural degree of separation between the margins of the two lids (Dalrymple's sign). Normally, when vision is directed

Fig. 368.—Exophthalmic goitre.

downwards, the upper lid moves concordantly with it. In this disease the upper lid follows tardily or not at all (von Graefe's sign): this symptom is not always present and may occur in other forms of exophthalmos. There is diminished frequency of winking and imperfect closure of the lids during the act (Stellwag's sign). There may be imperfect power of convergence (Möbius' sign), and often the skin of the eyelids shows pigmentation. Ophthalmoscopically veins and arteries may be somewhat distended, but specific signs are absent. One or more of the cardinal symptoms may be absent. The eye may become dislocated forwards between the lids, the orbicularis contracting in spasm behind it. Reduction is

effected by separating the lids and bringing them forwards over the eye, if necessary after blocking the facial nerve with novocain.

The disease is associated with over-activity of the thyroid gland, but the cause of the exophthalmos is unknown. It does not seem to be directly associated with over-producton of thyroid secretion or the degree of thyrotoxicosis, but may be due to an increased output of thyrotropic hormone from the anterior lobe of the pituitary, stimulated perhaps by the hypothalamus. Exophthalmos is not produced by over-treatment with thyroid extracts or thyroid hormone.

Paralysis of extrinsic ocular muscles, usually the external rectus, sometimes precedes the protrusion of the eyes (*exophthalmic ophthalmoplegia*). In these cases thyrotoxicosis seems to be absent, since the thyroid is not generally enlarged and basal metabolism is normal or subnormal. The disorder sometimes comes on after partial thyroidectomy, and may be aggravated by injections of thyrotropic pituitary hormone. The disease is progressive; diplopia persists, and the muscles may be pale, œdematous, and swollen to six times their normal size. More benign cases of paresis associated with exophthalmos, however, also occur.

It is necessary for the ophthalmic surgeon to be able to recognise the disease. Further details of its usual course and treatment must be sought in medical text-books.

**Pulsating Exophthalmos** is generally due to arterio-venous aneurysm, the communication taking place between the internal carotid artery and the cavernous sinus. The eyeball is protruded and the blood vessels of the conjunctiva and lids are widely dilated. The angular vein and its branches near the inner canthus are very prominent, and they can be seen, or more easily felt, to pulsate synchronously with the arterial pulse, since, owing to the arterio-venous communication, they are under arterial pressure. The patient complains of continual rumbling, as of a waterfall, and this can be heard on auscultation over the eye or orbit by the surgeon. The proptosis is diminished by steady pressure on the globe, and may be diminished or abrogated by pressure on the common carotid artery of the same side, or sometimes only by pressure on the carotid of the opposite side. Ophthalmoscopically the veins of the retina are greatly distended: there may be papillœdema with defective vision, which may amount to complete blindness. There is often considerable pain from stretching of the branches of the fifth nerve.

The cause of the arterio-venous aneurysm is usually a severe blow or fall upon the head, and is therefore commoner in men, but probably in all cases the walls of the artery are already degenerated. It may occur from syphilitic or other arteriosclerosis, without discoverable traumatism, especially when it occurs in women. The exophthalmos in rare cases subsides spontaneously. More commonly it increases, and may end in hæmorrhage or death from cerebral causes.

*Treatment.* Continuous pressure applied to the carotid artery which stops the pulsation usually fails to effect a cure. Ligature of the carotid has been more successful, but recurrence of pulsation not infrequently occurs. Ligature of both internal and external carotid does not appear to give better results. The opposite carotid may also be tied, but this should not be done for some weeks after the first operation, owing to risk to life from cerebral anæmia. This procedure also may fail to relieve the condition, and in these cases the distended veins have been dissected out, an operation of considerable danger.

**Intermittent Exophthalmos** occasionally occurs, generally when the head is depressed, enophthalmos not infrequently being present in the erect position. The proptosis is increased by pressure on the corresponding jugular vein. It is ascribed to varicosity of the orbital veins.

# SECTION VIII

## PREVENTIVE OPHTHALMOLOGY

### CHAPTER XXXIV

### The Causes and Prevention of Blindness

THE previous chapters have dealt chiefly with the diagnosis and treatment of already established diseases of the eye. An equally important branch of medical science is concerned with the prevention of disease, and although this aspect of ophthalmology has hitherto received less attention than it merits, it ought not to be ignored by the medical student or practitioner.

The most disastrous result of ocular disease, short of the relatively rare loss of life, is blindness. A study of the causes of blindness will enable the student to form a judgment as to the comparative danger of various ocular diseases.

The term "blindness" implies inability to perceive light; but it is obvious that many people who yet retain some slight degree of visual capacity are helpless from the economic standpoint. The Advisory Committee on the Welfare of the Blind, therefore, included among the blind all those who are "too blind to perform work for which eyesight is essential." The Register of the Blind for England and Wales, compiled on this basis, shows that there were 67,521 blind persons in those areas in 1935, and 8298 in Scotland in 1934. Some 70 per cent. of the total blind population are over the age of fifty, and 80 per cent. of blind persons are unemployable.

The factors producing blindness have a different rate of incidence at different ages. Thus, in a home for blind *infants*, Harman found about 50 per cent. due to ophthalmia neonatorum, 11 per cent. to intraocular inflammations, and 30 per cent. to congenital defects. Statistics of *children of school age* show that 20—30 per cent. were blind from ophthalmia neonatorum, 10—20 per cent. from interstitial keratitis, and 15—20 per cent. had optic atrophy due to various causes, including disseminated choroiditis. The statistics for *adults* are very unreliable, owing to defective case histories, the impossibility in many cases of determining the causes of blindness from examination of the patients, and other

reasons. In middle life the high incidence of ophthalmia neonatorum is still noticeable, choroiditis and optic atrophy are important, while iritis and iridocyclitis are markedly advanced in relative position. Many of these are symptomatic conditions, the underlying cause being often syphilis. Myopia is a prominent factor, accidents assume a high proportion, and glaucoma appears, increasing considerably in later life.

The importance of *ophthalmia neonatorum* as a cause of blindness is so great that it has been deemed advisable to discuss the measures for its prevention earlier in this book (*see* p. 161). While there is little evidence of any reduction in the incidence of this disease treatment by penicillin should lead to a reduction in the amount of blindness caused by it.

*Syphilis*, both in the congenital and in the acquired form, is responsible for a large amount of blindness. Harman found definite signs of congenital syphilis in one-third of a group of 1,855 blind children, and in most of these cases congenital syphilis was the undoubted cause of the blindness. At least 10—15 per cent. of cases of blindness in adults are probably due to syphilis, and these figures do not include cases due to vascular disease of possible syphilitic origin.

*Phlyctenular keratitis* was found to be the cause of blindness in 3·56 per cent. of 1,855 blind children (Harman). This disease and such conditions as blepharo-conjunctivitis are largely due to insanitary conditions of life. They might probably be eliminated as causes of blindness by the adequate provision for the education and treatment of the children in special residential schools, a method which has proved eminently successful for trachomatous children. *Measles* is another not uncommon cause of blindness through corneal ulceration; it emphasises the importance of proper treatment of the eyes by the medical practitioner. *Trachoma* (*q.v.*) is, fortunately, now a rare cause of blindness in this country.

*Myopia* was the cause of blindness in 3 per cent. of 1,855 blind children (Harman), and in 14 per cent. of 601 blind persons of all ages (Harman). These figures underestimate the serious distress and economic loss due to this cause. It is generally agreed that myopia is increased by near work but the tendency has been to exaggerate this influence (*vide* Chap. XXIV.). Special "myope" classes have been instituted for the education of short-sighted children. "It is convenient to classify myopia in two divisions—a 'school' myopia and a 'pernicious' myopia. The former is, as a rule, low in degree, does not progress beyond a certain extent, and is but rarely associated with other ocular changes. The fact that ophthalmic surgeons recognise a school myopia is the strongest argument for its prevention by the provision of all those measures which are summed up in the term

school hygiene'; such as good lighting in classrooms, good print in books, regulated needlework, a maximum of oral instruction, and, above all, the early correction of errors of refraction and the special supervision of those who show signs of being or becoming short-sighted. But it is the pernicious myopia which figures in these tables of blindness or partial blindness—a variety which commences at an early age, may progress rapidly, is very liable to be associated with serious intraocular disease, and therefore necessitates the provision of special methods for the safe education of the subjects of it. The cardinal aim should be to endeavour to prevent the continued development of the disease in the children affected; such prevention requires the complete elimination of any form of eye strain such as is involved in close work, and the provision of educational facilities under the best hygienic conditions, such as is aimed at in the myope classes." There is evidence that habitual close eyework, such as that of clerks, sempstresses, compositors, &c., if habitually undertaken in poor illumination and poor hygienic conditions, is harmful to the eyesight of myopes, and every effort should be made to direct these people into suitable occupations or, alternatively, into satisfactory environmental conditions from the point of view of visual hygiene.

*Glaucoma* is a serious factor in the production of blindness after middle life; in a home for the aged blind the percentage of such cases was 29·82. "Cases of glaucoma frequently come in the first instance under the cognisance of the general practitioner. The acute form is apt to be mistaken for iritis, with disastrous results if the treatment appropriate to this latter disease is adopted; on the other hand, as the disease is often ushered in by severe headache and vomiting, the essential ocular condition may be overlooked. and the case regarded as a simple 'bilious attack.' The chronic form is so insidious in its onset that it may easily be overlooked." It is unfortunately by no means rare to meet with cases of acute or chronic glaucoma which have been treated with atropine or allowed to progress without proper treatment. It is of the utmost importance that the student should pay special attention to this disease, and especially to the diagnostic features which distinguish it from iritis (*vide* p. 261). If there is the slightest doubt as to the presence of glaucoma the case should be referred *immediately* to an ophthalmic surgeon.

*Industrial conditions* may cause blindness, either by disease or accident. The chief diseases, such as poisons (lead, derivatives of benzene, &c.), glass- and iron-workers' cataract, miners' nystagmus, &c., have already been dealt with. Blindness due to industrial accidents is commonest among miners and in the engineering trades. Of 5,575 blind persons of all ages the blindness was due to industrial accidents in 7·2 per cent. (Scottish Register

of the Blind, 1922). More striking is the enormous economic loss entailed by relatively minor accidents, such as foreign bodies in the eye (*vide* p. 432), &c. Many of these could be entirely prevented by the use of appropriate guards, screens and goggles. These matters require special attention from factory medical officers; but of far greater importance to the ordinary medical practitioner is the grave risk of blindness due to sympathetic ophthalmia produced by penetrating injuries of the eye. "The danger of the development of sympathetic ophthalmia should always be present in the mind of the medical practitioner, and all cases of penetrating wounds of the eye should be placed immediately under the observation of an ophthalmic surgeon; in no eye condition is prompt co-operation between the general practitioner and the expert more essential."

(Further information and recommendations referring to the subject of this chapter will be found in the Report of the Departmental Committee of the Ministry of Health on the Causes and Prevention of Blindness. H.M. Stationery Office, 1922: and the Report on the Prevention of Blindness, Union of Counties Associations for the Blind, 1936.)

# CHAPTER XXXV

## The Hygiene of Vision

APART from the conditions which seriously endanger the eyesight, discussed briefly in the last chapter, there are many others which are liable to impair the efficiency of vision or the health of the individual. It is well known that the use of the eyes with uncorrected errors of refraction or muscle balance, or under unsuitable conditions of illumination, &c., cause ocular pain and discomfort (commonly known as "eye-strain"), headaches, migraine, and general malaise. More serious disorders and diseases have been attributed by some to these causes. The exact pathology of "eye-strain" is unknown, and the *rationale* of visual fatigue in the production of ocular and systematic disorders is largely a matter of conjecture. It is, in the first place, a safe principle to make the ametropic eye approximate by artificial means as nearly as possible to the emmetropic eye. This is effected by suitable spectacles. In the next place it is necessary to study the normal limits of adaptability of the eye to various conditions of illumination, &c., and to use the knowledge thus obtained to prevent these limits being transgressed. When we bear in mind the evolution of the visual apparatus in man, and the immense increase in the amount and nature of the work which it is called upon to perform in modern civilised life, it is surprising that eyes are capable of withstanding the strain.

*Errors of Refraction.* The correction of ametropia by glasses has already been discussed. It is evident that theoretically this correction should be made as early as possible, and especially before the increased strain of school life is encountered. Much advance has been made in this direction in recent years, and the routine examination of the eyes of young school children ensures the discovery of serious errors. Facilities for their correction and for the supply of suitable glasses are now prevalent. The most difficult problem in this connection is that of myopia, which has already been dealt with in Chap. XXXIV.

*Illumination.* Normal vision is capable of adaptation to very wide ranges of intensity and quality of illumination. Form vision is very defective under dark adaptation and with low intensity of illumination. As the intensity is increased and the eye becomes light-adapted visual acuity increases—rapidly at first, and then

only very slowly. The increase is proportional to the logarithm of the intensity of the illumination, so that successive doublings or treblings of the illumination cause only equal arithmetic increments of visual acuity as estimated by the distance at which a standard letter (1·25 mm. square) can be read. Above 10 foot-candles the increment becomes progressively less. For ordinary work an illumination of 5 foot-candles suffices, but for fine work much higher values are desirable (*vide* p. 686). At extremely high illuminations, visual acuity is diminished owing to glare. It has been found that discrimination is increased by using monochromatic light, chiefly owing to the elimination of chromatic aberration in the eye; it is best with yellow light, the brightest part of the photopic spectrum.

There are many factors, however, which influence visual acuity besides the intensity of the light. Among these is the size of the pupils, but more important is the amount and character of the light falling upon peripheral areas of the retina. Thus, it is undesirable that there should be too great contrast between the areas under observation and surrounding areas; *e.g.*, self-luminous figures with radio-active paint are very difficult to focus in complete darkness, especially in condition of fatigue. A brilliantly illuminated field of work in an otherwise dark room causes rapid alterations of adaptation which are deleterious. Hence a moderate amount of general illumination is preferable, and this has the additional advantage that it prevents the formation of very sharply defined shadows. On the other hand, it is very important that there should be no glaring lights in the field of vision; such lights should be carefully shaded. Care, too, should be taken to avoid direct reflection of light into the eyes. Thus, in reading, especially books written on shiny paper, and in working on bright metals, &c., if the source of light is in front of the eyes light is reflected directly into them. This light is useless for visual purposes, and indeed diminishes the contrasts which are the basis of discrimination. Hence the source of light should be placed laterally, and preferably to the left-hand side and somewhat behind the worker. Flickering lights should be avoided.

Various sources of light differ much in intensity and quality. The natural criterion is sunlight, which we are accustomed to regard as white light. Sunlight differs much, however, in "whiteness," and in intensity on different days, at different times of the day, and whether direct or diffuse. Owing to the adaptability of the eye it is difficult to judge the intensity of a given illumination. Measurements show that bright direct sunlight may give several thousand foot-candles, and an illumination of several hundred foot-candles on a well-placed desk is quite common. One

great advantage of daylight is its diffusion; the illumination of a room usually comes, not directly from the sun, but from a considerable area of sky, and is reinforced by innumerable reflections from buildings and other objects. Sunlight is much richer in luminous radiation of short wave length—blue and violet—than any artificial illuminants. Most modern illuminants have continuous spectra derived from incandescent solids; the higher the temperature the more nearly the energy distribution of the spectrum approximates to that of sunlight. An approximation to diffuse daylight for purposes of matching colours, etc., can be obtained by suitable filters (" daylight lamps ") and by fluorescent tubes. Incandescent gases—such as used in the mercury-vapour lamp—have line spectra; they therefore more nearly approximate monochromatic light.

Glare may be regarded as light in the wrong place. The more concentrated the light the more disturbing is the effect. Glare, therefore, varies rather with the intrinsic brilliancy of the light than with its intensity. Intrinsic brilliancy is defined as candle-power per square inch. Clear sky has a very low intrinsic brilliancy —about the same as the candle, viz., 2·5 candles per square inch. A metal filament has an intrinsic brilliancy of 800 c. per square inch, and an arc light 20,000 c. per square inch. The ratio of the intrinsic brilliancy of a source of light to that of the surrounding field should not exceed 100. In general, the eye works best when the object regarded is surrounded by a field illuminated to the same or slightly less degree. The illumination of the field must on no account be higher than that of the object. Glare is diminished in artificial interior illumination by the use of indirect lighting. In this method the light is reflected from the ceiling and suitably curved cornices, so that no direct light reaches the eye. By it shadows are almost eliminated. It is a restful, but monotonous mode of illumination; it is quite unsuited for certain purposes. Thus, sewing is very difficult with it, especially the sewing of monochromatic material, because the threads of the texture throw no shadows, and consequently their discrimination is made very difficult. In semi-indirect lighting the use of opalescent bowls permits of a certain amount of direct illumination.

Many modern illuminants emit a considerable amount of ultra-violet radiation, which may be deleterious (*vide* pp. 188, 373). Most of this is absorbed by glass, so that the dangers arising from this cause are slight and have been much over-rated. It must be remembered, however, that globes also absorb an appreciable amount of the luminous energy; even clear glass globes absorb 5—15 per cent., and opal globes as much as 10—40 per cent. The distribution of light from artificial sources varies greatly. It can be

modified by the use of reflectors and prismatic (holophane) globes. Too little attention has hitherto been paid by architects and others to the position and characters of light sources from the hygienic point of view. It is of great importance in the lighting of factories and workshops, and especially in that of schools. There has been great improvement in the lighting of schools, factories, shops, streets and houses of recent years, largely due to the work of the Illuminating Engineering Society, which has issued a schedule of Recommended Values of Illumination. The following list gives the general principles upon which they are based :—

| | Recommended Foot-candle Value. | Class of Task. |
|---|---|---|
| 1. | Above 50 | Precision work to a high degree of accuracy; tasks requiring rapid discrimination; displays. |
| 2. | 25—50 | Severe and prolonged visual tasks; discrimination or inspection of fine details of low contrast. |
| 3. | 15—25 | Prolonged critical visual tasks, such as proof reading, fine assembling, and fine machine work. |
| 4. | 10—15 | Visual tasks such as skilled benchwork, sustained reading and sewing on light goods. |
| 5. | 6—10 | Less exacting visual tasks, such as casual reading and large assembly work. |
| 6. | 4—6 | Work of simple character not involving close attention to fine detail. |
| 7. | 2—4 | Casual observation where no specific work is performed. |

*Reading and Writing.* Considering the vast importance of reading and writing in modern life it is surprising that they have been so little investigated by physiologists and ophthalmologists. The forms of printed types are derived from manuscripts, and have been modified for technical reasons. Further advance has been almost entirely empirical, and even in the best presses more care has been exercised in obtaining æsthetic effects than in fostering legibility.

If we consider ordinary Roman printed characters we find that all capital letters extend above the line. Of the small letters thirteen are short, eight extend above the line (ascending letters), and four below the line (descending letters). There are thus twice as many ascending as descending letters, and in an ordinary page of print it will be found that of the long letters about 85 per cent. are ascending and only 15 per cent. descending. Examination of the short letters shows that their most characteristic features are in the upper parts. Hence, in reading, attention is specially directed to the upper parts of the letters, as is strikingly

demonstrated by covering the lower parts of a line of print with a card. The print is almost as legible as if it were uncovered. If, however, the upper halves of the letters are covered, it is almost, if not quite, impossible to read the print.

The ends of the lines of which letters are composed are commonly emphasised by means of serifs. These were doubtless introduced empirically, but the advantage in sharpness of definition has a physiological basis. They counteract irradiation, and hence the visibility of letters is improved if the serifs are triangular.

The tendency of typefounders has been to minimise the differences between letters, probably with a view to greater regularity of line and uniformity in appearance. For example, round letters have been flattened laterally and square letters rounded. The loops of b, d, p, and q, have been equalised to o. If the lower parts of short letters are covered, the similarity in the topmost curves of a, c, e, o, s, of n and r, of h and b, or of n and p, is much greater in modern print than in some early samples.

Legibility is not determined solely by visibility in the physiological sense of the term. Thus, the emphasis of some lines in letters, which originated in the use of reeds and pens for writing, increases legibility whilst diminishing visibility. A child learning to read depends upon physiological visibility; hence there should be little difference between the breadth of the thick and slender strokes. As facility in reading is acquired, legibility is increased by diminishing the breadth of the slender strokes, and as smaller letters are used the diminution must be more rapid than that of the heavy strokes, so that the interspaces may not be unduly contracted. At the same time, the slender strokes must not transgress the limits of visibility at reading distance, and their distribution should be emphasised by suitably formed serifs. Hence, Jaeger small types are more legible than Snellen's.

The spacing of the letters and words has a considerable effect upon legibility. Irradiation plays an important part here. Roughly speaking, the interspace between letters should be at least as broad as the blanks in m or n, but round letters like o and e should have slightly less interspace than square letters. Owing to irradiation the interspaces in general look larger than they really are, and two o's separated by a space look farther apart than two n's separated by the same space. Javal attributes a large part of the "remarkable legibility of English books" to the shortness of most English words and the consequent multiplication of blank interspaces. Of course, the spacing of words, and to a less degree of letters, in ordinary printing is very largely haphazard as far as legibility is concerned, the main object of the printer being to obtain general uniformity of

appearance with rigid equality in the lengths of the lines. There is some difference of opinion as to whether "leading" or interlinear spacing is beneficial. Owing to the design of the blocks of type there is always a small space between the lower limits of descending and the upper limits of ascending letters, even without leading.

A line of print is read in a series of small jumps. At each pause a group of about ten letters is more or less accurately visualised; the movements are too rapid to permit of visualisation whilst they are occurring. The number of leaps taken by the eye remains the same irrespective of the distance of the book, so long as this is consistent with legibility. A child reading makes more jumps in a line than the average, and the same applies to people reading a foreign language or correcting proofs. Attention is directed chiefly to the commencements of words, and words are not read by letters but by their general configuration. There is, therefore, a very important psychological factor involved in the act of reading, quite apart from the interpretation of the meaning of the words.

Enough has been said to show that reading is a highly complex act, and the rules which can at present be devised for the avoidance of strain and discomfort involve a multiplicity of factors which have not yet been satisfactorily correlated.

*Handicrafts.* The same visual principles as have been discussed above underlie the carrying on of many handicrafts and industrial processes, but each provides specific problems. For some types of very fine work convex lenses bringing the near point to 8 or 9 inches from the eye, combined with appropriate prisms, bases in, magnify the retinal images and have been found to give much relief.

No attempt has been made in this Section to deal exhaustively with so extensive a subject as Preventive Ophthalmology, but it has been deemed advisable to indicate to the student how innumerable and complex are the applications of ophthalmology to everyday life.

# APPENDIX I

## PRELIMINARY INVESTIGATION OF THE PATIENT

SCIENTIFIC observation can only be fostered by methodical investigation. The student is recommended to study each patient according to the following scheme :—

(1) **General condition of the patient.**

(2) **Position of the head.** Characteristic in paralyses of extraocular muscles (*vide* p. 556).

(3) **Face.** Note asymmetry, facial paralysis, affections of the skin, *e.g.*, herpes ophthalmicus, &c.

(4) **Position of the eyebrows.** Vicarious action of the frontalis in ptosis (*vide* p. 638), &c.

(5) **Orbits.** Exophthalmos, enophthalmos, &c.

(6) **Eyeballs.** (*a*) Position and direction—strabismus.

(*b*) Movements (*vide* p. 551).

(*c*) Size and shape—microphthalmia, glaucoma (*vide* p. 285); myopia (*vide* p. 521), buphthalmia (*vide* p. 302), staphyloma, &c.

(7) **Lids.** (*a*) Position—ptosis, ectropion, entropion, &c.

(*b*) Palpebral aperture—ptosis, exophthalmos, &c.

(*c*) Movements—ptosis, exophthalmic goitre, &c.

(*d*) Margins—blepharitis, tumours, &c.

(*e*) Lashes—trichiasis, distichiasis, &c.

(*f*) Glands—hordeolum, chalazion, &c.

(*g*) Puncta lacrymalia—eversion, occlusion, &c.

(*h*) Lacrymal sac—swelling, regurgitation, &c.

(8) **Conjunctiva.** (*a*) Ocular — congestion (conjunctival and ciliary), secretion, phlyctenules, growths, wounds, &c.

(*b*) Palpebral—congestion, granulations, scars, concretions, ulcers, &c.

(*c*) Plica semilunaris—displacement in pterygium, growths, &c.

(*d*) Caruncle—inflammation, granulations, growths, &c.

(9) **Cornea.** (*a*) Size—glaucoma, buphthalmia, &c.

(*b*) Curvature—conical, buphthalmia, anterior staphyloma, abrasions (*vide* p. 85), &c.

(*c*) Surface—corneal reflex, abrasions, ulcers, foreign bodies, &c.

(*d*) Transparency—ulcers, nebulæ, keratitis (superficial and deep vascularisation, *vide* p. 90), "k.p.," striate opacity, &c.

(10) **Sclerotic.** (*a*) Curvature and colour—myopia, staphyloma, episcleritis, &c.

(*b*) Vessels—ciliary injection, episcleritis, scleritis, &c.

(11) **Anterior chamber.** (*a*) Depth—($\alpha$) shallow—perforating wound, glaucoma, dislocation of lens, &c.; ($\beta$) deep—buphthalmia, iridocyclitis, dislocation of lens, &c.; ($\gamma$) irregular—iris bombé, dislocation of lens, &c.

(*b*) Contents—cloudy aqueous, hypopyon, hyphæma, foreign bodies, dislocated lens, &c.

(12) **Iris.** (*a*) Colour—muddy in iritis, heterochromia (congenital and in iridocyclitis), atrophy, ectropion of uvea, melanomata, &c.

(*b*) Position, especially plane of surface—iris bombé, retraction in iridocyclitis, pseudo-glioma, &c.

(13) **Pupils.** (*a*) Relative size (*vide* p. 92).

(*b*) Reaction to light—direct, consensual, maintenance of constriction (*vide* p. 93).

(*c*) Reaction on convergence.

(*d*) Synechiæ—anterior and posterior.

(14) **Intraocular tension.** Increased in glaucoma, iridocyclitis; diminished in iridocyclitis. perforation or rupture of the globe, &c.

(15) **Central vision** (*vide* p. 130).

(16) **Lens.** By oblique illumination and the ophthalmoscope.

(17) **Vitreous.** Opacities, fluidity, foreign bodies, hæmorrhage, "retinitis proliferans," persistent hyaloid artery, &c.

(18) **Fundus.** (*a*) Optic disc—blurring of the edges, swelling, cupping, colour, crescents, &c.

(*b*) Retinal vessels—size, contour, tortuosity, &c.

(*c*) General view—retinitis, choroiditis, &c.

(*d*) Periphery.

(*e*) Macula.

(19) **Field of vision.**

# APPENDIX II

## THERAPEUTIC NOTES

*(The strengths of lotions, &c., are given in percentages: 1 per cent. = gr. v. to ʒ i. (approximately))*

### Lids

*Lotions.* 3 per cent. Sodium bicarbonate.
3 per cent. Borax.

These are used for dissolving the crusts in blepharitis. They may be used in conjunction with 1 to 2 per cent. salicylic acid, 1 to 2 per cent. resorcin, liquor carbonis detergens (♏ i.—iv. to ʒ i.), &c.

*Pigments.* 10 per cent. Silver nitrate.
1 per cent. Picric acid.
1 per cent. Salicylic acid.

These are used for ulcerative blepharitis; the excess should be removed with cotton wool.

Liquor Tinctorium.

This consists of equal parts of crystal violet (0·5 per cent.) and brilliant green (0·5 per cent.) in equal parts of alcohol and water.

*Ointments.* 2 per cent. Ammoniated mercury.
3 per cent. Yellow oxide of mercury.

These are well rubbed into the lashes for five minutes three times a day after removing the crusts in ulcerative blepharitis. Alternative preparations are 1 per cent. salicylic acid, 3 per cent. airol, 2 per cent. resorcin, 3 per cent. ichthyol, &c.

Triple dye Jelly.

This consists of gentian violet 1/400, brilliant green 1/400, and neutral acriflavine 1/1000. It is used for burns of the lids (*v.* p. 643).

### Conjunctiva

*Lotions.* 3 per cent. Boric acid.
1 in 5000 Perchloride of mercury.
1 in 8000 Oxycyanide of mercury.
1 per cent. Mercurochrome.
1 in 1500 Acriflavine.
1 in 2500 Metaphen.
1 in 2500 Hexyl-resorcin.

These are used as cleansing lotions in acute conjunctivitis, and have slight antiseptic properties; they should be mixed with an equal quantity of hot water as a rule. Mercury compounds occasionally cause severe dermatitis.

2 per cent. Borax.

This, combined with dilute hydrocyanic acid (1 per cent.), relieves irritation in mild conjunctivitis.

1 per cent. Alum.

¼ per cent. Zinc sulphate or chloride.

These are astringent lotions used in chronic conjunctivitis. Zinc lotion is specially indicated in angular conjunctivitis (*q.v.*). Alternative preparations are 1 per cent. tannin, ½ per cent. copper sulphate, 3 to 6 per cent. sodium sozoiodol, ½ per cent. zinc sulphocarbolate, &c.

1 per cent. Quinine hydrochloride or sulphate.

This is recommended for membranous conjunctivitis. The least possible amount of dilute sulphuric acid should be used to dissolve the salt if the sulphate is used.

*Pigments.* 2 per cent. Silver nitrate.

This is used in acute conjunctivitis. Alternative preparations are 10 to 40 per cent. argyrol, 10 to 40 per cent. protargol, 5 to 10 per cent. argentamin, &c.; these are probably not so effectual (*vide* p. 154).

2 to 4 per cent. Perchloride of mercury in glycerin and water.

This is occasionally used in trachoma; it must not be allowed to touch the cornea. Iced compresses should be applied immediately after the application is made.

*Drops.* The astringent lotions may be used in the form of drops. Silver nitrate should not be ordered in this form on account of the staining which may occur from prolonged use.

2 to 4 per cent. Cocaine hydrochloride.

This is used for producing local anæsthesia. Conjunctival and corneal anæsthesia is complete after instilling four drops at intervals of five minutes. During the intervals the eyes should be kept closed on account of the desiccating action on the corneal epithelium. In intraocular operations one drop should be instilled into the opposite eye (*vide* p. 474). It must not be used for hypodermic injection, but must be replaced by the less toxic novocain.

1 per cent. Pantocain.

This derivative of novocain has largely replaced the use of cocaine, 2 per cent. solution giving an even stronger and more prolonged anæsthesia than 4 per cent. cocaine. It does not dilate the pupil nor dull the corneal epithelium.

2 per cent. β-Eucain hydrochloride.

2 per cent. Alypin.

These may be used instead of cocaine. They do not dilate the pupil and are less toxic than cocaine, but they cause considerable smarting.

1 to 10 per cent. Dionin.

This causes intense œdema of the conjunctiva; after prolonged use the reaction is slight or absent. The patient complains of a burning sensation which quickly disappears. The first application should be made by the surgeon. The drug stimulates the lymph flow, and has therefore been recommended in a large variety of cases, *e.g.*, corneal nebulæ, scleritis, iridocyclitis, &c. It must be used with caution.

*Subconjunctival Injections.*—These have been recommended for the same

reason as dionin; and their value, if any, is still unproved. Many solutions have been used. Sterile 2 to 10 per cent. salt solution is the best; the others probably possess no advantage, and are in some cases liable to cause necrosis. The injection is made with a hypodermic needle under the bulbar conjunctiva as far back as possible above the globe. There is considerable reaction and pain. Not more than 5 to 10 minims should be injected every other day.

*Ointments.* Vaseline.

15 per cent. Boric acid ointment.

These are used to prevent the lids from sticking together and thus causing retention of secretion.

1 to 3 per cent. Yellow oxide of mercury.

This is used as a stimulant and antiseptic in phlyctenular and chronic conjunctivitis.

### Cornea

*Lotions.* The same collyria as for conjunctival conditions are used as cleansing and antiseptic measures. The astringent collyria are seldom indicated.

*Pigments.* 1 per cent. Silver nitrate.

This is indicated in some cases of marginal ulceration (*vide* p. 223).

Pure carbolic acid.

This is used as a cauterising agent in hypopyon ulcer (*vide* p. 217).

*Drops.* These are the ordinary mydriatics and cycloplegics, and in rare cases miotics (*vide infra*).

Dionin or subconjunctival hypertonic saline injection is sometimes used with a view to clearing corneal nebulæ. Two per cent. fluorescein in 3 per cent. bicarbonate of sodium solution is used for staining ulcers and abrasions for diagnostic purposes.

*Ointments.* These are the same as for conjunctival conditions.

2 to 5 per cent. iodoform, xeroform, or airol ointment may be used in corneal ulceration. Dionin (5 to 10 per cent.) and mydriatics may be prescribed in ointment form. 1 to 5 per cent. yellow oxide of mercury ointment is used in gradually increasing strength to aid in the clearing of nebulæ and the opacity of interstitial keratitis; it may be combined with dionin.

### Mydriatics and Cycloplegics; Miotics

*Mydriatics and Cycloplegics* (*vide* p. 62). ½ to 1 per cent. Atropine sulphate. ¼ to ½ per cent. Hyoscine or Scopolamine hydrobromide. ¼ to ½ per cent. Duboisine sulphate. ½ to 1 per cent. Daturine sulphate.

These are used as drops or ointment in corneal ulcers, iritis, iridocyclitis, &c. Atropine is used generally, the others being employed as substitutes when atropine causes irritation. Hyoscine occasionally causes delirium and should be watched.

1 per cent. Homatropine hydrobromide, with or without 2 per cent. cocaine hydrochloride.

1 to 2 per cent. Euphthalmine hydrochloride.

These are used for investigation of refraction and for ophthalmoscopic examination; also for the diagnosis of synechiæ (*vide* p. 260) and occasionally for the diagnosis of increased tension (*vide* p. 260). For refraction solutions of the same strength of the bases in castor oil are more certain in their action.

1 per cent. Paredrine.

This dilates the pupil with very slight effect on accommodation.

Lævoglaucosan is a more powerful mydriatic than atropine.

It consists of 2 per cent. lævo-rotatory deltanephrin with 2 per cent. methylaminoacetopyrocatechol. Two drops are repeatedly instilled at intervals of fifteen minutes. It is an expensive drug.

2 per cent. Cocaine hydrochloride.

This is used occasionally in old people as a mydriatic for ophthalmoscopic purposes, being less likely to raise the tension, the effect being readily counteracted by miotics.

"Mydricain." The most powerful mydriatic effect has been obtained by subconjunctival injection of a mixture of atropine, cocaine and suprarenin.

Each 5 minim dose of "mydricain" consists of atropine sulphate gr. 1/60, cocaine hydrochloride gr. 1/10, and lævo-rotatory suprarenin gr. 1/600, with sodium chloride gr. 1/80, and chlorbutol gr. 1/120, in sterilised water. (Flynn, *Brit. Jl. of Ophthal.*, XVII., p. 298. 1933.)

*Miotics* (*vide* p. 63). ½ to 1 per cent. Pilocarpine hydrochloride. ¼ to 1 per cent. Eserine or Physostigmine sulphate or salicylate, with or without 1 per cent. cocaine hydrochloride.

These are used in glaucoma (*q.v.*), and occasionally in other cases (*vide* p. 223). The effect of cocaine is to assist the absorption of the other drug.

0·75 per cent. "Doryl" (carbaminoyl cholin) is more active than 2 per cent. pilocarpine and less active than 1 per cent. eserine, and may be used as a substitute for these miotics.

### Hot Bathings

Much better than the usual hot fomentations is the method of hot bathing used at Moorfields Eye Hospital. A pad of cotton wool is tied into the bowl of a wooden spoon. The wool is dipped into a bowl of boiling water, and is then approximated to the closed eye. As soon as it has cooled sufficiently it is brought into contact with the closed lids. As soon as it ceases to feel hot the wool is again dipped in the hot water and the process repeated. The bathing is continued for ten to fifteen minutes, and then a pad of dry warm cotton wool is bandaged over the eye. The hot bathings may be repeated frequently.

## The Treatment of Syphilitic Affections

In cases of suspected syphilitic disease of the eye the possibility of demonstrating the presence of the spirochæte should be borne in mind. More generally useful is the application of Wassermann's test, for which the aid of a bacteriologist is advisable. No anti-syphilitic remedies should be used until the test has been applied. When the lesion is probably syphilitic the patient should be brought rapidly under the influence of penicillin administered parenterally (*vide infra*), mercury and iodine or arsenic compounds. Mercury may be given by inunctions, intramuscular injections, or intravenous injections, supplemented if necessary by administration by the mouth. Inunction is generally employed, and for this purpose the oleate of mercury is preferable to the ordinary mercury ointment. Intramuscular and intravenous injections require special technique. Intravenous injection of salvarsan ("606") N.A.B., or its equivalent is sometimes remarkably successful, especially in the acute stage of syphilitic manifestations. Substitutes for the ordinary iodides are sajodin or iodoglidine tablets (1 to 3, *i.e.*, 7 to 15 gr. three times a day), iodipin (30 gr. of 25 per cent. = about 10 gr. of potassium iodide), &c.

## Chemotherapy

The advances in chemotherapy in recent years have completely revolutionised our therapeusis in acute infections. Two potent agents are now in common use in the sulphonamide group of drugs and penicillin. The exact mode of action of neither of these is known but both are bacteriostatic rather than bacteriocidal. The result is that since the organisms are inhibited from growing and multiplying, the natural defences of the body can deal easily with those already present. As soon as the influence of the drug is withdrawn, the remaining bacteria can resume growth and multiplication, so that the rationale of treatment is to keep the drug continuously in contact with the infected tissue until the infection is overcome. Since these drugs are rapidly excreted from the body or diffuse from any site of local application, repeated or continuous administration during this crucial period is necessary.

## The Sulphonamides

Several drugs belonging to this group are now in general use, the newer compounds having the general advantage of being less liable to cause toxic effects than the original sulphanilamide (prontosil). They are effective in a large number of infections, particularly those caused by streptococci, gonococci, staphylococci, pneumococci, meningococci and other organisms as indicated in the text. From the ophthalmological point of view they may be given orally or locally.

*Systematic administration.* In their systemic administration an attempt should be made to obtain the maximum concentration in the blood of 10 mgm. per 100 c.c. over a period sufficient to allow the infection to be overcome. Theoretically the dosage should be calculated for each patient on a basis of body weight, but in practice in the average adult an initial dose of 2·0 gm. followed by a 6 hourly maintenance dose of 1·0 gm. is suitable. For a newly born infant doses 1/10 of this are indicated.

The drugs in common use belonging to this group are the original sulphanilamide, sulphapyridine, sulphathiazole, sulphadiazine and sulphamezathine. Of these the last three are less toxic in their general effects and more easily borne by the patient, as well as being more effective against the organisms of ophthalmological importance than sulphanilamide. They all diffuse into the eye although there is evidence that the penetration of

sulphathaizole into the ocular tissues is somewhat limited. Their clinical use is applicable to conjunctival, orbital and intra-ocular infections caused by bacteria which are susceptible to them. There is also evidence that they are effective against the group of large size viruses which appear to be responsible for trachoma and blenorrhœa of the new-born.

*Local use.* The local use of sulphonamides has largely been replaced by that of penicillin; nevertheless they have some application. The most effective for general application because of its complete lack of irritability is sodium sulphacetamide (albucid) which may be employed as a solution in drops (30 per cent.) or as an ointment (2·5 or 6 per cent.).

## Penicillin

Penicillin is an extraordinarily effective bacteriostatic agent to which Gram positive organisms and Gram negative cocci are sensitive; the Gram negative bacilli as a class are relatively insensitive to it. It is, however, a large moleculed substance and, unlike the sulphonamides, cannot diffuse into the eye in effective quantity unless administered in very high concentration. While the sulphonamides are therefore indicated in intra-ocular infections, penicillin is most generally useful in extra-ocular infections. In deep-seated inflammations of the orbit or lids it is administered parenterally; in superficial inflammations of the conjunctiva and cornea it is administered locally. In addition to being a more potent bacteriocidal agent than the sulphonamides it has the great advantage of being completely non-toxic.

*Systemic administration.* To maintain an effective concentration of penicillin in the tissues a dose of the order of 100,000 units should be administered daily for some days. The only infection which appears to be cured in 24 hours is gonorrhœa. Since the drug is destroyed by the acids in the stomach oral administration has so far been ineffective. The common method of administration is intra-muscular injections, 15,000 units dissolved in 2 c.c. normal saline or distilled water being injected unremittingly at 3-hourly intervals. An alternative method is by a continuous intra-muscular drip (100,000 units in 100 c.c. saline per day), but probably the most useful is a daily intra-muscular injection of 100,000 or more units in oil, the drug being continuously absorbed during the 24 hours.

*Local administration.* Penicillin may be given in the form of drops, ointment or powder or as subconjunctival injections. Drops should contain from 500 to 2,500 units per c.c. and should be instilled into the eye at frequent intervals depending on the acuteness of the infection. In the early stages of an acute infection, intervals varying from a minute to a quarter of an hour may be indicated; in less acute infections intervals of two to three hours will be sufficient. The drops tend to lose their potency in a few days' time, so that only fresh solutions should be employed, the life of which is prolonged by keeping in the cold. At 5° C. ordinary solutions will retain their activity for 3 days: but if used in the pure form potency is retained much longer. They are most easily made up in the fresh state from tablets of sodium penicillin dissolved in distilled water. An ointment may be made on a basis of 30 per cent. lanette wax in water at a strength of 250 units or more per gm. or in pure vaseline. An alternative is the official B.P. method whereby the calcium salt is incorporated in a basis of yellow soft paraffin and wool fat. The ointment produces a less potent but more prolonged effect and is more liable to cause an irritation of the lids than the drops; it maintains its potency for several weeks.

*Powder.* For use in powder form dry calcium penicillin is diluted with one of the sulphonamide powders (sulphathiazole) to give a concentration of from 1,000 to 5,000 units per gm. In powder form penicillin retains its

potency for many months at a low temperature. Such a powder can be used in the treatment of corneal ulcers but is most effective in the treatment of an infected socket or dusted into an open wound of the lids or orbit.

*Intra-ocular administration.* The recent introduction of pure penicillin has allowed its use in sufficient concentration to admit of therapeutic levels being reached and maintained in the eye, since in the pure form local irritation is practically absent. The most convenient method of administration is by subconjunctival injections of upwards of 10,000 units in distilled water repeated every 3 hours. In desperate cases of intra-ocular suppuration the solution may be injected directly into the anterior chamber or the vitreous: for this purpose the drug must be absolutely pure, or an intense and sometimes destructive reaction may follow.

## Serum and Vaccine Treatment

In diphtheritic conjunctivitis antitoxic serum must be used. The indications for other sera and vaccines in the domain of ophthalmology are much less precise. Antipneumococcic serum for the treatment of hypopyon ulcer has proved disappointing. Antigonococcic serum has been advocated in ophthalmia neonatorum and gonorrhœal ophthalmia. In desperate cases of septic infection of the eye after perforating wound, accidental or operative, a polyvalent serum may be employed, or if possible a vaccine made from a culture taken from the eye: oral administration of antidiphtheritic serum has been recommended in these cases. The vaccine treatment of tuberculosis is much used. Many obscure pathological conditions in the eye suggest the possibility of a tuberculous origin. Diagnosis may be facilitated in some, probably a minority of cases, by the application of von Pirquet's cutaneous test. Wolff-Eisner and Calmette's conjunctival reaction should not be used. Occasionally, as in conjunctival tuberculosis, it is possible to place the diagnosis beyond doubt by inoculation experiments on animals; the incubation period, however, is lengthy (20 to 30 days). Tuberculin treatment is best carried out by a bacteriologist familiar with the technique.

## Shock Therapy

Subcutaneous injection of foreign proteins usually produces considerable febrile reaction and sometimes improvement in obscure or recalcitrant inflammatory eye diseases. Milk is generally used. It should be boiled twice for 4 minutes each time. The initial dose is 5 c.c. injected intramuscularly; the dose may be increased to 10 or 12 c.c. (1 c.c. for infants under 1 year, 2 c.c. up to 5 years and 3 c.c. up to 10 years of age). Three or four doses are given at 2- or 3-day intervals. It is a wise precaution to give a preliminary injection of 1 c.c. to guard against anaphylactic shock. Typhoid-paratyphoid vaccine is also used intravenously and has been effective in sympathetic ophthalmia. Antidiphtheritic serum is good, but the danger of anaphylactic shock on repetition must be guarded against.

The general reaction in shock therapy is at first low temperature with rigor, slow pulse and nausea; followed by high temperature, rapid pulse and leucocytosis. There is local erythema and tenderness. The focal reaction is shown by hyperæmia and inflammatory changes, accompanied by relief of pain.

## Vitamins

The following are the vitamins most important in ophthalmology, showing their natural sources, daily need, functions, and deficiency effects.

**A.** (Higher alcohol synthesised from carotene in the liver). *Carotene* from carrots, green vegetables; **A** from fat fish, esp. liver—*cod liver oil, halibut*

*oil*—egg yolk, milk: *carotene* 3 mgms; **A.** 3,000 units: maintenance of healthy ectodermal structures (respiratory, alimentary, urinary, *conjunctival, corneal, retinal*); *production of visual purple.* Deficiency effects—dermatoses, demyelination (Vth nerve and C.N.S.), diminished resistance to infections; *xerosis, xerophthalmia, keratomalacia, night blindness, ? cataract.*

**$B_1$** (Thiamin or aneurin, a pyramidine thiazole compound). From all foodstuffs, esp. lean pork, beans, peas, nuts, *whole* grain and flour, beef, *yeast:* 1 mgm.: carbohydrate metabolism. Deficiency effects—beri-beri, peripheral neuritis; *corneal and conjunctival dystrophy, retrobulbar neuritis.*

**$B_2$** or **G.** (Ribo- or lacto-flavin.) Same sources as $B_1$: 1 mgm.: formation of yellow enzyme of Warburg. Deficiency effects—glossitis and cheilosis; *vascularizing keratitis, ? cataract.*

**C.** (Ascorbic or cevitamic acid). From fresh fruit and vegetables (destroyed by heating): 50 mgms.: Blood formation, osteogenesis, *lens metabolism.* Deficiency effects—scurvy, anæmia, osteoplasia; *conjunctival and retinal hæmorrhages, keratoconjunctivitis.*

**D.** (Calciferol—isomer of ergosterol, formed by ultraviolet light). From animal fats, esp. *cod liver oil and halibut oil;* sunshine: 1,000 units, 0·025 mgm.: Ca and P metabolism. Deficiency effects—rickets, osteomalacia, dental caries, tetany; *cataract, ? myopia.*

**K.** (Dimethylnaphthoquinone). From alfalfa: prothrombin formation.

**P.** (Flavone). From citrous fruits: maintenance of health of capillaries. Deficiency effects of K and P—hæmorrhagic conditions.

These vitamins have been isolated or synthesised, and are available in proprietary preparations. Deficiency should be counteracted as far as possible in the diet. Little harm seems to accrue from large doses, except in the case of calciferol from hypercalcæmia.

## Care of Instruments

Ophthalmic instruments should be kept in an air-tight glass cabinet, or when not constantly in use in velvet-lined cases.

All instruments should be sterilised before use by boiling in 3 per cent. sodium carbonate solution (*not* bicarbonate), made with *distilled* water. This procedure does not impair the cutting edges, but knives and scissors should not be boiled more than three to five minutes: this is amply sufficient if the surfaces are bright and free from tarnish, as they ought to be. If distilled water cannot be obtained the cutting instruments should be well soaked in pure carbolic acid before being transferred to the dish.

The instruments should be removed from the steriliser *immediately* before operating and used dry. *In no case must instruments be immersed in boric lotion,* since it tarnishes the steel.

It is, however, much safer to use the instruments dry. It is almost impossible to sterilise the skin of the hands efficiently, and if the instruments are wet fluid from the fingers is liable to run along them into the eye. The points of knives, &c., should be dipped in sterile saline immediately before use to facilitate their passage through the tissues.

The surgeon should wear a sterilised gown and also a mask containing a layer of cellophane covering the nose and mouth, for all intraocular operations.

# APPENDIX III

## REQUIREMENTS OF CANDIDATES FOR ADMISSION INTO THE PUBLIC SERVICES

### National Service Grading

#### Eyes (Diseases of the Eye and Defects of Vision)

*Diseases of the Eye.* Men suffering from diseases of the eye, as distinct from defects of visual acuity, will be referred for special examination and graded in accordance with the results. In cases of acute disease with a good prospect of recovery grading will be deferred for the requisite time.

*Visual Standards.* Men will be classified, so far as their visual acuity is concerned, according to the following standards :—

*Standard* 1. Unaided vision is not less than 6/6 in one eye and not less than 6/9 in the other.

*Standard* 2. Unaided vision is less than in Standard 1 but is either not less than 6/12 in each eye or is not less than 6/6 in the right eye, and not less than 6/36 in the left eye.

*Standard* 3. Unaided vision is less than in Standard 2 but vision can be corrected to at least Standard 2.

*Note.* In those cases where unaided vision is below 6/60 in either eye the men will be referred to an ophthalmologist and where a myopia of more than minus 7 in any meridian is found he will be placed in Standard 7.

*Standard* 4. Unaided vision is less than in Standard 2 and vision cannot be corrected to Standard 2 but can be corrected to at least 6/12 in one eye and to at least 6/36 in the other.

*Note.* This standard includes those men whose left eye is the " master " eye and whose vision with or without correction is not less than 6/12 in the left eye, and not less than 6/36 in the right eye.

*Standard* 5. The conditions in Standards 1 to 4 cannot be attained but vision can be corrected to at least 6/24 in each eye.

*Standard* 6. Vision in one eye, with or without glasses, is not less than 6/12 and in the other is less than 6/36 with or without glasses, or has been lost or practically lost and investigation as to the cause of the loss is satisfactory.

*Standard* 7. Vision is below Standards 1 to 6.

*Grading.* Men whose visual acuity is in Standard 1 to 4 will, so far as eyesight is concerned, be placed in Grade I and those whose visual acuity is in Standards 5 and 6 in Grade II.

*Reference to Ophthalmologists.* In addition to those men already mentioned in the note to Standard 3, all men who cannot be placed in Standards 1, 2 or 3 will be referred to an ophthalmologist, unless the board is satisfied that any improvement in vision which might be obtained by the provision of glasses, or by an alteration of the glasses which a man may be wearing, would not affect classification.

## ENTRANTS INTO THE ARMY

The vision of candidates for Commissions or recruits for the Army must belong to standards 1-6 in the National Service grading (*vide supra*).

## ENTRANTS INTO THE ROYAL NAVY

### ENTRY STANDARDS OF VISION, OFFICERS R.N.

**CADETS, DARTMOUTH.**

| Distant Vision. | | Near Vision. | Colour Vision. |
|---|---|---|---|
| 6/6 | 6/6 | Snellen, D = 0·5 (Jaeger, 1) | Grade I. |

(*a*) Limit of hypermetropia permissible (under homatropine).

*In the better eye.*

Hypermetropia, 1·5 dioptre. Simple hypermetropic astigmatism, 0·75 dioptre. Compound hypermetropic astigmatism: the error in the more hypermetropic meridian must not exceed 1·5 dioptre of which not more than 0·75 dioptre may be due to astigmatism.

*In the worse eye.*

Hypermetropia, 2·5 dioptres. Simple hypermetropic astigmatism, 1·0 dioptre. Compound hypermetropic astigmatism: 2·5 dioptres in the meridian of greater error, of which not more than 1·0 dioptre may be due to astigmatism.

(*b*) Fields of vision to be normal.

(*c*) Glasses are not allowed on duty.

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**CADETS, EXECUTIVE. ("Special Entry.")**

| Distant Vision. | | Near Vision. | Colour Vision. |
|---|---|---|---|
| 6/6 | 6/12 | Snellen, D = 0·5 (Jaeger, 1) | Grade I. |

(*a*), (*b*), (*c*) As for Cadets, Dartmouth.

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**CADETS, ENGINEERING BRANCH. ("Special Entry.")**

| Distant Vision. | | Near Vision. | Colour Vision. |
|---|---|---|---|
| 6/9 | 6/12 | Snellen, D = 0·5 (Jaeger, 1) | Grade II. |

(*a*) Hypermetropia exceeding 5·0 dioptres (under homatropine) in the meridian of greater error will disqualify.

(*b*), (*c*) As for Cadets, Dartmouth.

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**PAYMASTER CADETS. ("Special Entry.")**

| Distant Vision. | | Near Vision. | Colour Vision. |
|---|---|---|---|
| 6/60 | 6/60 | Snellen, D = 0·6 (Jaeger, 2) with glasses. | Grade III. |

(*a*) As for Cadets, Engineering Branch.

(*b*) Fields of vision to be normal.

(*c*) Glasses permitted on duty. Distant vision, with glasses, to be not less than 6/6 in one eye and 6/24 in the other eye.

| | Distant Vision. | Near Vision. | Colour Vision. |
|---|---|---|---|
| ROYAL MARINES. | 6/12 6/12 | Snellen, D = 0·5 (Jaeger, 1) | Grade II. |
| | (*a*) As for Cadets, Engineering Branch. | | |
| | (*b*) (*c*) As for Cadets, Dartmouth. | | |

| | Distant Vision. | Near Vision. | Colour Vision. |
|---|---|---|---|
| MEDICAL OFFICERS, DENTAL OFFICERS, NURSING SISTERS. | 6/60 6/60 | Snellen, D = 0·6 (Jaeger, 2) with glasses. | Grade II. |
| | (*a*) As for Paymaster Cadets. | | |
| | (*b*) A gross defect will disqualify. | | |
| | (*c*) As for Paymaster Cadets. | | |

| | Distant Vision. | Near Vision. | Colour Vision. |
|---|---|---|---|
| INSTRUCTOR OFFICERS, SCHOOLMASTERS, CHAPLAINS. | 6/60 6/60 | Snellen, D = 0·6 (Jaeger, 2) with glasses. | Grade III. |
| | (*a*) (*b*) (*c*) As for Medical Officers. | | |

Squint, deformity or any chronic disease of the eyes or eyelids will disqualify for entry into any Branch.

The standard of distant vision must be attained without glasses, and, except where otherwise stated, visual acuity, with glasses, must be not lower than 6/6 in each eye tested separately. For certain Branches (noted above), glasses may be used during the test of near vision.

*Colour Vision.* Details regarding testing and grading will be found in Medical Research Council, Special Report Series, No. 185 (H.M. Stationery Office). Candidates for Branches in which the wearing of glasses on duty is permitted may use them for the colour vision test.

## THE ROYAL NAVY

### EYESIGHT STANDARDS FOR MEN

The vision of all candidates for entry, re-entry, or re-engagement, is to be tested without glasses, unless otherwise stated.

| | Entry Standard. | | Colour Vision in all Periods. |
|---|---|---|---|
| | Distant. | Near. | |
| Seamen (entered as boys) | 6/6—6/6 | D = 0·5 | I |
| Seamen (special service) | 6/9—6/9 | D = 0·5 | I |
| Signalmen (entered as boys). | 6/6—6/6 | D = 0·5 | I |
| Signalmen (special service. | 6/6—6/6 | D = 0·5 | I |
| W/T ratings (entered as boys). | 6/6—6/6 | D = 0·5 | I |

| | Entry Standard. | | Colour Vision in all Periods. |
|---|---|---|---|
| | Distant. | Near. | |
| W/T ratings (special service). | 6/9—6/12 | D = 0·6 | II |
| Stokers . . . | 6/9—6/9 | D = 0·6 | II |
| Stoker Petty Officers Mechanicians. | 6/9 both eyes together, worse eye not less than 6/18. | D = 0·6 | II |
| E.R.A. entered as Apprentice. | 6/9—6/9 | D = 0·5 | II |
| *E.R.A. direct entry . | 6/9 both eyes together, worse eye not less than 6/8. | D = 0·6 | |
| E.A. entered as Apprentice. | 6/9—6/9 | D = 0·5 | II |
| *E.A. direct entry . | 6/12 both eyes together, worse eye not less than 6/24. | D = 0·6 | |
| O.A. entered as Apprentice. | 6/9—6/9 | D = 0·5 | II |
| *O.A. direct entry . | 6/12 both eyes together, worse eye not less than 6/24. | D = 0·6 | |
| Shipwright entered as Apprentice. | 6/9—6/9 | D = 0·5 | II |
| Shipwright direct entry | 6/12 both eyes together, worse eye not less than 6/24. | D = 0·6 | |
| Cooper, Blacksmith, Plumber, Joiner, Painter. | 6/12 both eyes together, worse eye not less than 6/24 | D = 0·6 | II |
| Regulating Branch . | On entry into Branch, if in first period, 6/9—6/12. | On entry into Branch, if in first period, D = 0·6, without glasses. | II |

* Candidates for direct entry as Artificers, who are required to pass a trade test before entry, are not to be rejected for defective colour vision provided the defect is not sufficient to prevent them performing their technical duties.

| | Entry Standard. | | Colour Vision in all Periods. |
|---|---|---|---|
| | Distant | Near. | |
| Sick Berth Staff . . | 6/12 both eyes together, worse eye not less than 6/24. | D = 0·6 | II |
| Writers, Supply ratings, Stewards, Cooks. | 6/12 both eyes together, worse eye not less than 6/24. | D = 0·6 | III |
| Royal Marines . . | 6/9—6/9 | D = 0·5 | II |
| Royal Marine Bandsmen | 6/12—6/12 | D = 0·6 | II |
| Sailmakers . . . | On entry into Branch if in first period. 6/12—6/12 | D = 0·6 without glasses. | — |
| Photographer Ratings . | 6/12 both eyes together, worse eye not less than 6/24. | D = 0·6 | II |

W.T. = Wireless Telegraphist.
E.A. = Electrical Artificer.
E.R.A. = Engine Room Artificer.
O.A. = Ordnance Artificer.

## The Royal Air Force

FLYING PERSONNEL must have unaided vision in each eye of not less than 6/18 which must be correctable to 6/6 in each eye with spectacles, provided the refraction error in any meridian does not exceed ± 2·5 dioptres Both eyes must have good fields of vision; colour vision must be normal; binocular fusion and convergence must be good and there must not be more than 4 prism dioptres of esophoria or exophoria or 1 prism dioptre of hyperphoria.

### Notes

*Hypermetropia* is ruled out because of the associated upset of convergence on sustained efforts of accommodation, and because of the tendency of a failure of accommodation to develop at high altitudes.

*Muscle Balance Tests.* These are important from the point of view of landing, since judgment of distance in the air depends entirely on the visual sense divorced from the usual environment in contact with the ground. It has been shown that exophorics tend to flatten out their aircraft too early, judging the ground to be nearer than it actually is, while esophorics are inclined to fly into the ground.

Four tests are employed in the following order:—

(*a*) The convergence test.
(*b*) The cover test.
(*c*) The red-green test.
(*d*) The Bishop Harman diaphragm test.

### (*a*) The Convergence Test

The test is carried out as follows: Hold a pencil about a foot from the eyes of the subject under examination; see that the point is accurately between the eyes and on a level with the root of the nose. Tell him to fix the point and then move it directly towards him, observing what happens to the eyes as they converge. The following results may occur:—

(*a*) Both eyes may converge fixing the pencil until it gets within an inch or two from the root of the nose, indicating no want of convergence. Record distance in inches.

(*b*) One or other eye ceases to fix the object and may wander out, or both eyes fail to keep up fixation. The subject may even resist by throwing back his head and complain either that it hurts him to follow the pencil or that he sees double. In such cases, convergence is defective. Record in inches the distance at which such lack of convergence manifests itself.

Convergence power is estimated by giving the following values:—2 inches or under, very good, possibly excessive; 2 to 3 inches good; 3 to 4 inches, fair; 4 inches and over, poor.

### (*b*) The Cover Test

Hold a pencil in front of the subject at about 1 foot from the nose, asking him to look at the print, then cover one eye with a card. Move the pencil from side to side, bringing it finally to the centre and uncovering. Repeat the test for the other eye in a similar manner.

A perfectly balanced pair of eyes will remain fixed on the pencil whether one is uncovered or covered, whereas movement inwards or outwards on uncovering ("latency") shows some lack of balance. Where the eyes are perfectly balanced on uncovering, record as "lat., nil."

Where "latency" (lat.) is found, the degree of movement, convergent (con.) or divergent (div.), and the speed of return to normal alignment is important. A movement of less than 30 degrees is recorded as "slight" (sl.), or more than this as "marked" (mkd.). The return to normal alignment, "recovery" (rec.), may be "rapid" (rpd.), "moderate" (mod.), "slow" (sl.), or "by stages," *i.e.*, jerky, and should be recorded as such. In some cases there may be an appreciable interval before recovery starts, this should be noted as "lag." or there may be no recovery of alignment until the eye is again directed to the object (*e.g.*, by covering the other eye). Record this as "Rec. nil."

Record no movement or latency, *i.e.*, perfect balance as "lat. nil." Record movement as "Al or mkd. lat. div. or con." Recovery therefrom as "Rapid, mod., slow, by stages, or nil."

### (*c*) The Red-Green Test

In a black box containing a lamp, a vertical slot $\frac{3}{8}$ inch wide by 5 inches long, is glazed in the upper $2\frac{1}{2}$ inches with red and in the lower $2\frac{1}{2}$ inches with green. The person under examination—wearing a pair of reversible frames containing red glass in one eyepiece and green in the other—is told to look at the slot illuminated from behind and asked to state what he sees:—

1. (*a*) The two lights in their proper position (*i.e.*, alignment of visual axes and correct ocular muscle balance); (*b*) definite displacement up or down and/or right or left (*i.e.*, hyper- or hypo- and/or eso- or exo-phoria); (*c*) one or both may wander from original position and back again (*i.e.*, tendency to aphoria, but with some or full power of correction).

2. At first two lights; then an occasional disappearance of one of them, either the same one all the time, or the two alternately (*i.e.*, mastery of one eye, either continuous or alternating, with neglect of vision by the other).
3. Either colour singly, but never the two together (*i.e.*, alternate perception of the object with suppression of the image of the other eye).

(It may happen that the two colours will be superimposed and fused into one image; reversal of the frame obviates this difficulty, as the colours will then be separated.) The nature and amount of displacement indicate the quality and quantity of the heterophoria thus revealed. A true exophoric case may give temporary esophoric displacement owing to over-compensation, but, as fatigue sets in, the image passes through the orthophoric to the exophoric position.

### (*d*) The Bishop Harman Diaphragm Test

The instrument consists of a rod, one end of which carries an endpiece shaped to fit the upper lip, to which it may be pressed by means of a handle fixed below the rod. At the other end, 44 cm. away, is a vertical card-holder in the plane at right angles to the rod. Between these, 11 cm. from, and parallel to, the card-holder, is a diaphragm in which a rectangular aperture is cut, through which the subject views the test card. The aperture is capable of being widened or narrowed at will by means of two shutters, one on either side of the opening, worked by a right-and-left-handed screw, which is rotated by means of a milled head. Movement of the shutters operates a pointer registering on a quadrant scale, which can itself be adjusted to the widely varying pupillary distances of individual subjects. With wide-open diaphragm, all the figures on the test-card—say 1, 2, 3, 4, 5, 6, 7—can be seen by both eyes together, but as the edges of the diaphragm move towards each other, this binocular overlay becomes more and more reduced, until finally there remains nothing in sight which is common to the two eyes.

In performing the test, the subject is asked to state precisely what happens to the figures when the size of the aperture is gradually diminished. For instance, he may state that :—

(*a*) The figures at one or other end of the card begin to disappear—indicating suppression of one or other image.
(*b*) The middle figures tend to crowd together and mix up—indicating esophoria.
(*c*) The figures divide, and the centre figure tends to duplicate or a black bar appears between them—indicating exophoria.
(*d*) Some of the figures deviate to a higher or lower level—indicating hypophoria or hyperphoria.
(*e*) A black bar appears, obliterating the middle figure, but the rest remain in the proper relative positions—indicating that the overlap has been entirely cut down, and that there is a negative gap which is represented by blackness; in such an instance the reading will be below zero, and will indicate a perfect control of balance.

It has been found that a reading of "3" on the arbitrary scale is a "border-line" one, and that "5" is bad. First-class "landers" usually give a reading of zero or below; average "landers" zero to "2"; doubtful "landers" "2" to "3"; whilst those giving readings of "3" to "5" or more fall generally into the category of bad "landers."

*The Visual Fields.* Both eyes must have good fields of vision as tested by hand movements. Normal fields of vision in both eyes are necessary for flying, because :—

(i.) The pilot requires to obtain the widest possible view of aircraft and other objects in his proximity.

(ii.) He depends mainly upon the vision of the peripheral fields for judgment of the pace of his aircraft in relation to laterally placed stationary objects.

(iii.) The grey horizon (false horizon) at night is best perceived by the rod elements of the peripheral fields.

(iv.) There are certain ocular diseases which reveal alterations in the peripheral fields, while normal central vision is retained; to detect these, the rough test by hand movement is, generally speaking, quite sufficient.

*Colour Vision* This must be normal, or safe, as normal colour perception is requisite in navigation and landing, owing to the use of coloured lights as signals.

(*a*) *Colour defective, safe.*

Anomalous Trichromaticism can be accepted. When tested by the Ishihara colour plates such a candidate must read accurately plates 1, 7, 9, 12 and 13. The plates will be found numbered in small print below the colour scheme.

(*b*) *Colour defective, unsafe.*

Candidates who misname the colours red and green in any combination on the lantern or who fail to name accurately the Ishihara charts noted above are rejected.

GROUND PERSONNEL—OFFICERS AND W.A.A.F. OFFICERS AND MEMBERS OF P.M., R.A.F.N.S., OTHER THAN THOSE SPECIALLY REFERRED TO BELOW.

1. (*a*) *Visual Acuity.* The lowest limit of visual acuity that may be accepted is 3/60 in each eye, provided it is correctable to at least 6/12 in each eye; or one eye may be blind* or lost, but the acuity in the other eye must not be worse than 6/60, correctable to 6/9 at least. The latter may be accepted only on the recommendation of an Ophthalmic Specialist, and only then in cases where the remaining eye contains no important errors of refraction and no pathological state, *e.g.*, chronic conjunctivitis, or old corneal ulcer.

(*b*) *Colour Vision.* May be unsafe, but is to be recorded.

(*c*) *Special Assessment.* All candidates for Commissions in A. and S.D. Branch must in addition to the assessment of fitness for that branch, be marked "Fit (or unfit) Code and Cypher Duties." (See para. 2.)

2. *Officers and W.A.A.F. Officers (A. and S.D.) for Code and Cypher Duties.*

(*a*) *Visual Acuity.* The lowest acceptable standard is 6/60 in each eye correctable to 6/6 in each eye.

(*b*) *Convergence.* This must be at least to 4 inches (10 cm.).

(*c*) *Colour Vision.* May be unsafe, but is to be recorded.

(*d*) *General.* No disease of the eyelids likely to become chronic, or of media or retinæ, producing a visual impairment to constitute a cause for failure in efficient performance of these duties, should be present. The history of a squint, a tendency to undue ocular fatigue or recurrent headache is a cause of rejection for these duties.

3. (*a*) *W.A.A.F. Officers—Photo Interpretation Duties.* 6/24 or better in each eye without glasses, correctable to 6/6. Hypermetropia not to exceed + 2·5D. Sph. Convergence 4 inches or better.

* One-eyed personnel; these will be referred for special examination.*

4. (*b*) *Wireless Operator, Radio Telephone Operator, W/T Morse (slip reader) Operator, Teleprinter Operator.*

(i) *Visual Acuity.* Either 6/60 or better in each eye, correctable to 6/6 in one eye; or worse than 6/60 in one eye, or blindness in it, or loss of it, provided investigation into cause is satisfactory, and not less than 6/12 in the other eye, correctable to 6/6. (See also above.)

(ii) *Colour Vision.* This need not be tested.

(iii) *Other Ocular Requirements.* Chronic disease of the eyes or eyelids, even though quiescent, is a cause of permanent rejection.

*Note.*—For Radio Telephone Operators on G.C.I. sets, the standards of visual acuity are the same as Radio Operators.

5. (*c*) *Radio Operator.*

(i) *Visual Acuity.* 6/60 or better in each eye, correctable to 6/6 in each eye.

(ii) *Colour Vision.* Must be normal or safe.

(iii) *Other Ocular Requirements.*

(*a*) No chronic disease of the eyes or eyelids, even though quiescent, may be present. If myopia is present, the fundi must be healthy.

(*b*) Hypermetropia must not exceed + 3 D. sphere.

(*c*) Convergence must be at least to 4 inches (10 cm.).

(*d*) Cover test must show a good result.

(*e*) A history of squint, a tendency to undue ocular fatigue or recurrent headache is a cause for rejection.

## Civil Flying Certificates

The tests in general are as for the Royal Air Force, but for private flying ("A" Licence), and for navigators ("B" Licence), a certain allowance is made and correcting glasses are allowed to be worn provided a degree of visual acuity can be attained, with or without glasses, equal to at least 80 per cent. of the normal visual acuity for each eye taken separately, or 90 per cent. for one eye and 70 per cent. for the other.

The same standard *without* glasses holds for pilots for "B" Licence. (*Note:* Visual acuity is equal to 100 per cent. normal when at a given distance—20 feet in Great Britain—the letters of the standard type subtend an angle of five minutes. The easiest way to test 80 per cent. normal vision is to get the applicant to read standard $\frac{6}{6}$ type at a distance of 16 feet. Similarly, 90 per cent. vision is obtained with $\frac{6}{6}$ type at 18 feet and 70 per cent. at 14 feet. In other words, every 2 feet nearer the type represents a diminution of 10 per cent. in visual acuity.)

## Masters and Mates of the Mercantile Marine

### I.—Letter Test.

1. *Letter Test to be Passed First.*—The first test which the candidate is required to undergo is the letter test.

2. *Apparatus Used.*—The letter test to be used for all candidates is that conducted on Snellen's principle by means of sheets of letters.

3. *Standard of Vision Required.*—With the exceptions indicated below (*see* paragraph 6), every candidate will be required to read correctly nine of the twelve letters in the sixth line and eight of the fifteen letters in the seventh line of a test sheet placed in a good light at a distance of 16 feet from the eye.

4. *Method of Testing.*—The test sheets should be hung on the wall, *in a*

*good light, but not in direct sunlight*, at a height of five or six feet from the ground. The candidate should be placed at a distance of exactly 16 feet from the sheets, and exactly opposite them. This distance should be carefully measured, and should never in any circumstances be varied.

One of the sheets should then be exposed, and the candidate should be asked to read the letters on each sheet, beginning at the top and going downwards. Any mistakes which he makes should be carefully noted. If then it is found that he has read correctly at least nine letters in the sixth line and eight letters in the seventh line of a sheet, the candidate may be considered to have normal vision, and should be marked " passed " in the appropriate column of the form of application (Exn. 2 or Exn. 2A, as the case may be).

5. *Passing or Failure.*—If at the conclusion of the test the candidate is found to reach the required standard, he may be considered to have passed, and the Examiner should proceed with the lantern test, unless the candidate holds a certificate of competency. If the candidate fails to reach the standard required for the certificate entered for, he should be tested with at least four sheets, and the Examiner should fill in a Form Exn. 17b, the number of mistakes made in each line of each sheet, and explain to the candidate the alternatives mentioned in paragraph 31 (*d*).

Failure to pass the letter test is due to some defect in form vision, and the Board are advised that such defects are sometimes curable. Whenever, therefore, a candidate fails to pass this test the Examiner should advise him to consult an ophthalmic surgeon with a view to ascertaining what is the nature of the defect in his form vision, and whether it is curable.

6. *Lower Standard Required in Certain Cases.*—Candidates who are in possession of certificates obtained before January 1st, 1914, may be regarded as passing the letter test if they can read correctly with both eyes at least five of the eight letters in the fifth line of a test sheet.

7. *Tests to be Varied.*—The Examiner should take care, by varying the order of the test sheets and by every other means in his power, to guard against the possibility of any deception on the part of the candidate.

8. *Result of Examination to be Reported.*—The result of every examination in the letter test should be reported, in the case of a candidate for a certificate of competency, to the Registrar-General of Shipping and Seamen on Form Exn. 2, and to the Principal Examiner of Master and Mates on Form Exn. 14; and, in the case of a candidate for the sight tests only, to the Registrar-General of Shipping and Seamen on Form Exn. 2A.

## II.—Lantern Test.

9. *Apparatus.*—A special lantern and a mirror have been provided for this test. The lantern should be placed directly in front of the mirror, so that the front part of the lantern is exactly ten feet from the mirror. Care should be taken that the lantern is properly placed, that is to say, the lights reflected in the mirror must show clearly when viewed from the position of the Candidate on the left of the lantern. The Examiner should always satisfy himself that these conditions are fulfilled before commencing the examination.

10. *Darkness Adaptation.—It is essential that a Candidate should be kept in a room which is either completely or partially darkened for at least a quarter of an hour before he is required to undergo this test.*

Before the examination commences the Examiner must satisfy himself that the room in which it is conducted is so darkened as to exclude all daylight.

11. *Method of Testing.*—The lantern supplied for the examination is so constructed as to allow one large or two small lights to be visible, and is

fitted with 12 glasses of three colours—red, white and green. At the commencement of the examination the Examiner should show to the candidate a series of lights through the large aperture, and should require him to name the colours as they appear to him. Care should be taken in showing the white light to emphasise the fact that this light is not a pure white. If a Candidate makes a mistake of calling this light "red," a proper red light should be shown immediately after and the Candidate's attention directed to the difference between the two.

After a series of lights through the large aperture has been shown, the Examiner should make a complete circuit with the two small apertures, requiring the Candidate to name the colours of each set of two lights from left to right. To prevent any possibility of the order in which the lights are arranged from being learnt, the Examiner should at least twice in each circuit go back a varying number of colours.

A record of any mistakes made with either the large aperture or the two smaller apertures should be kept on Form Exn. 17b in accordance with the instructions thereon. In all such cases the mistakes made by a candidate in the letter test should also be recorded on the form.

12. *Passing or Failure.*—If a Candidate with either the large aperture or the two smaller apertures of the lantern mistakes red for green or green for red, he should be considered to have "failed" in the lantern test.

If the only mistake made by the Candidate with the lantern is to call the white light "red," and if after his attention has been specially directed to the difference between the two he makes no further mistake of this nature, he should be considered to have passed in the lantern test.

If a Candidate makes any other mistake with the lantern, *i.e.*, if he calls white "red" repeatedly or red "white" at all, or confuses green and white, his case should be submitted to the Principal Examiner of Masters and Mates and he should be told that the decision as to whether he is passed or failed, or must undergo a further examination, will be communicated to him in due course. Pending the receipt of the Principal Examiner's instructions such a Candidate should only be allowed to proceed with the remainder of the examination for a Certificate of Competency on the express understanding that the latter examination will be cancelled in the event of failure in the Sight Tests.

13. *Further Examination and Appeals.*—If in the cases covered by the preceding paragraph the Principal Examiner decides that a further examination is necessary, arrangements will be made for a special examination to be held in London.

If, however, on the report of the local Examiner the Principal Examiner decides that the nature of the mistakes made shows conclusively that a Candidate's sight is so defective as to render him unfit to hold a Certificate, the Candidate shall be considered to have failed.

In cases where, upon the report of the local Examiner, a Candidate is failed by the Principal Examiner, as well as in the cases covered by paragraph 12, the Board will be prepared to allow a Candidate who is dissatisfied with this decision to appeal for a special examination in London, subject to the conditions set out in paragraph 31 (*f*), (*g*) and (*h*).

## Candidates for Appointments in the Civil Service

Any serious defect of vision. A moderate degree of ordinary short sight corrected by glasses would not as a rule be regarded as a disqualification, but candidates for certain situations as, *e.g.*, preventive man in the Department of Customs and Excise, are liable to disqualification for defective colour vision or any other defect of vision. Candidates for some other appointments of a special character would be rejected for colour blindness,

but for the Covenanted Civil Service of India and for ordinary Home Appointments it is not by itself a disqualification.

*Candidates for the Civil Service Commissioners' certificate for posts in the Postal Service.*

No fixed standards of vision are prescribed. Each case is judged on its merits after the medical examination which every candidate must pass as a condition of certification.

Loss of sight of one eye by mechanical injury. If the other eye is sound and sufficient and not likely to become affected, the question of fitness will be specially considered; but for situations for which the rules lay down that "any serious defect of vision will disqualify," loss of sight of one eye would usually be regarded as a bar to appointment.

## Pilot Service

1. A candidate shall be examined both as to his physical fitness and as to his acuteness of vision and colour perception.

2. The sight tests shall be carried out in accordance with the Board of Trade Regulations applying to examinations for certificates of competency in force for the time being.

3. A candidate must have no defect of sight, and must have full normal vision in both eyes, each eye being examined separately without the aid of spectacles.

4. A candidate shall be disqualified by any imperfection of his colour sense or by squint, or any defective action of the eye muscles or any disease of the eye.

5. A candidate must be also otherwise physically fit for the duties of a pilot.

## Candidates for Scholarships and Teacherships under Local Education Authorities

The recommendations of the Council of British Ophthalmologists are as follows:—

*Visual Acuity.* In all candidates for scholarships and teacherships visual acuity, with correcting glasses, should not be less than $\frac{6}{9}$ in the better eye.

*Myopia.* A child of eleven with less than two dioptres of myopia should be passed. A child of eleven with two or three dioptres of myopia in either eye should be passed on probation and re-examined every six months

A child of eleven with three or more dioptres of myopia in the better eye should be rejected for scholarship training.

At the age of fifteen those with more than four dioptres of myopia in the better eye should be deemed unfit to train for the teaching profession.

Candidates for entry to training colleges, who are, as a rule, about eighteen years of age, if they have more than five dioptres of myopia in the better eye should be rejected.

*Astigmatism.* Simple myopic astigmatism exceeding three dioptres in the better eye should be a cause of rejection of candidates both for scholarships and for training as teachers. In cases of compound myopic astigmatism, unless the myopia is stationary, astigmatism of even two dioptres may be a cause of rejection.

Cases of hypermetropic astigmatism should be rejected only if visual acuity, with correcting glasses, in the better eye is less than $\frac{6}{9}$.

*Hypermetropia.* The defect should not be a cause of rejection unless vision, with correcting glasses, in the better eye is less than $\frac{6}{6}$.

*Special Cases.* If there is only one eye, or if there is only one useful

eye, the other being amblyopic from non-progressive disease, which in no way affects nor is likely to affect the good eye, the case should be judged on the condition of the good eye.

In the case of candidates for University and technical senior scholarships, or those intending to specialise as teachers of certain technical subjects, greater latitude may be allowed after consideration of special circumstances as to nature of work, condition of the candidate's eyes in other respects, etc.

## Board of Education

Report of Eyesight of Candidates for Teacherships by Ophthalmic Surgeon approved by Board. Information is required on the following points :—

1. The nature and extent of the defect.
2. Whether it can be compensated by glasses so as to enable that candidate to perform the duties of a teacher satisfactorily.
3. Whether it is progressive.
4. Whether the existence of the defect is likely either :
   (*a*) To shorten the term of active service as a teacher, or
   (*b*) To interfere with the candidate's efficiency as a teacher.

## Railway Servants

*Class A.* Drivers, Train motormen, Firemen and Engine-cleaners. On application to enter the service.

1. Form Vision Test. $\frac{6}{6}$ each eye separately.
2. Colour Vision Test. To pass the test by the Edridge-Green Colour Perception Lantern.

Re-examination :—

(*a*) After illness or accident considered likely to affect the sight, also, if considered desirable, after any alleged cases of mistaking signals.

(*b*) On promotion to Fireman, the standard required on re-examination being $\frac{6}{6}$ $\frac{9}{12}$, and $\frac{6}{6}$ together.

(*c*) On appointment as Driver, the standard required on re-examination being $\frac{6}{6}$ $\frac{6}{12}$, and $\frac{6}{6}$ together.

(*d*) Further re-examination will be made at 50, 55 and 60 years of age.

(*e*) If the examinee cannot pass under clause (*c*) but can reach $\frac{6}{12}$ $\frac{9}{18}$, and $\frac{6}{12}$ together, he must be re-examined every three years up to the age of 60.

(*f*) If an examinee fails to pass under clause (*e*) he shall be given practical tests by means of semaphore signals at 800 yards and 1,000 yards, by flags of the standard design at 400 and 500 yards, and by the usual colour vision test.

A man will be considered eligible for *shunting duties* who passes the signal test at 200 and 400 yards, and the flag test at 150 yards, and the usual colour vision test.

In all cases, when 60 years of age is reached and every two years thereafter, re-examination will take place, both in form and colour vision.

*Other Grades in Class A.* On application to enter the service.

1. Form Vision. $\frac{6}{6}$ each eye separately.
2. Colour Vision. Test as above.

Re-examination. As for Drivers, except that in the Practical Test the distance for signals is 200 and 400 yards, and for flags 150 yards.

The use of Glasses is permitted in the case of Signalmen, Passenger Guards and Ticket Collectors undergoing re-examination.

*Class B.* On application to enter the service.

1. Form Vision. $\frac{6}{9}$ $\frac{6}{12}$, and $\frac{6}{9}$ together.
2. Colour Vision. Test as above.

*Class C.* On application to enter the service.
1. Form Vision. $\frac{6}{18}$ each eye separately.
2. Colour Vision. No test.

N.B. The above regulations are subject to slight variations by individual Railway Companies.

## Police Force

There is no general standard of vision.

*Constables of the Metropolitan Police Force*

Form Vision. $\frac{6}{9}$ each eye separately.

## Metropolitan Police Regulations for Drivers of Motor Cabs Certificate of Vision

I hereby certify that I have this day examined by Snellen's test type the vision of.....an applicant for a Licence to act as Driver of.....with the following result:—

Acuity—R.E. without glasses..... L.E. without glasses.....
R.E. with glasses..... L.E. with glasses.....

Field of vision by hand test.....

He has no squint, colour blindness, or other defect of vision which would affect his fitness as such driver.

He is.....to act as driver of the before-mentioned class of public carriage.

Signature, etc.

Drivers of motor omnibuses, char-a-bancs and electrical tramway cars are not allowed optical aid to bring the vision up to the requisite standard.

## Motor Driver's Vision Test—Ministry of Transport's Requirements

The person signing a licence for driving a motor car is required to certify that he is able to read a stationary well-illuminated motor car number-plate at 25 yards distance. Since the figures on such a number-plate average 3½ inches high by 2½ inches wide, this standard of vision approximately equals a visual acuity of $\frac{6}{12}$.

## Form of Certificate for the Purpose of the Old Age Pensions Act, 1908, and the Blind Persons Act, 1920

I have examined the eyes of.....who stated that $\frac{\text{he}}{\text{she}}$ was a claimant to a pension under the Blind Persons Act, 1930, and I am of opinion that $\frac{\text{he}}{\text{she}}$ is .....*so blind as to be unable to perform any work for which eyesight is essential.

Signature.....

Qualifications.....

Date.....

Please insert the word "not" if you consider it necessary.

# INDEX

*(The principal page references are printed in heavier type.)*

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